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XI.

A STUDY IN TRANSILLUMINATION.*

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The first recorded use of sinus transillumination as a diagnostic measure is by Voltolini,[1] in 1888. Shortly before this time the incandescent lamp had been perfected to such an extent that it was found practical to hold in a patient's mouth a small bulb attached to a tongue depressor and to view the resulting translucency of the cheeks in a dark room. Using such a device, Voltolini reported findings which to him suggested that the patient was suffering either from an "empyema or a cyst of the antrum." The method was taken up in 1889 by Heryng,[2] who made further improvements in the electrical apparatus and expressed himself as favorably impressed with its use as a diagnostic measure. As in the case of any new procedure, however, opponents began to appear to throw doubt on the value of transillumination as a reliable clinical aid. Both Srebrny[3] and Ziem[4] reported unfavorable experiences in which transillumination gave negative results in spite of subsequent recovery of definite pus from the maxillary antrum. The former claimed that only when the sinus was completely filled would there be definite lack of translucency of the corresponding cheek. Moreover, it was soon recognized that other factors besides the fluid content of the sinus had a profound effect on the degree of translucency. Thus, anatomic variations in shape and size tended to render observations inaccurate, as did unequal densities of the surrounding facial bones. Finally, it became

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evident that in the standard technic of observing the relative translucency of the facial tissues there were variations in the light sense of different individuals and in their ability to detect small differences in translucency. It was in 1892 that Davidsohn[5] first clarified some of these points of contention and added certain observations which rendered transillumination more reliable. He called attention to the fact that when a source of light is placed in the mouth, not only is there produced a translucency by the passage of light rays through the normal antrum and soft tissues of the cheek, but that also there is observed an illumination of the pupil of the eye, owing to rays which pass through the antrum, the orbital plate and the fat-free areas near the front of the eyeball. He therefore suggested as the diagnostic sign this illumination of the pupil as preferable to that of the cheek. He did this as the result of experiments in which he artificially covered the floor of the maxillary antrum with an opaque medium, after which he noted that although, on transillumination, there was no illumination of the pupil, nevertheless the cheek tissues still radiated light. This he interpreted as being due to the fact that certain of the light rays from the source in the mouth, after passing through the palate, enter the nasal chamber on that side and thence, by reflection from the surfaces of the turbinates and septum, pass across the antrum and through the cheek. By blocking off first the inferior and then the middle meatus he was able to exclude these reflected rays and so prevent this source of translucency of the tissues of the cheek. He found, however, that these reflected rays from the nasal chamber did not produce any illumination of the pupil. Moreover, in the presence of abnormal thickening of the lateral nasal wall or presence of hypertrophies or polyposis of the turbinates, there was no reflection of light into the antrum. From the results of these studies Davidsohn concluded that so long as the illumination of the pupil is made use of as the diagnostic criterion, any amount of pus sufficient to interrupt the passage of light through the floor of the antrum will be sufficient to give a positive diagnostic result. If translucency of the cheek is taken as the diagnostic sign, then small amounts of an opaque fluid will not prevent the passage of light rays reflected from the nasal surface from giving a false impression of the absence of pathologic conditions. This situation undoubtedly explains the conflicting reports of results of the earliest investigators in this field. While Davidsohn was thus able to formulate the principle that if pus were present in the antrum, even in small amounts, the pupil would remain dark, he recognized that the darkness of the pupil did not by any means prove the presence of pus or other obstructing fluid, since any of the various anatomic abnormalities might

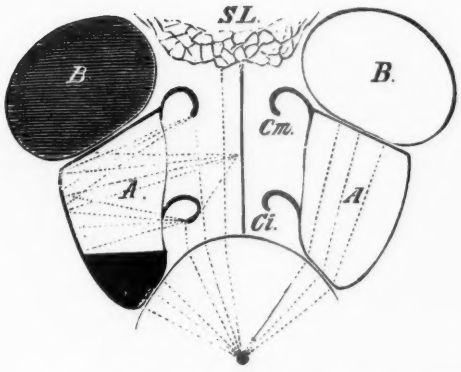

Fig. 1. Diagram showing the pathway of light during transillumination of the maxillary antrum. *B*, bulbus oculi. *A*, maxillary antrum. *Cm*, middle turbinate. *Ci*, inferior turbinate. (After Davidsohn.)

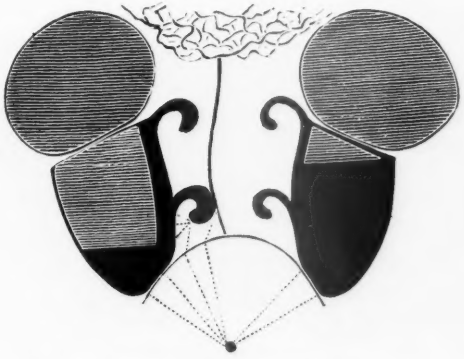

Fig. 2. Diagram showing interference with light rays by (left) fluid in the antrum and thickened nasal wall and (right) antrum full of fluid. (After Davidsohn.)

prevent light rays from reaching the pupil. Among these he stressed an abnormally high arched palate, an unusually narrow or laterally placed sinus or marked thickening of the peripheral bone. On the other hand, he maintained that if the pupil showed any evidence of illumination, the presence of pus was definitely excluded. In Figs. 1 and 2 are represented diagrammatically these principles of sinus transillumination as laid down by Davidsohn.

In Fig. 1, to the left, is shown the course of light rays emanating from a central source in the mouth and passing through an antrum containing a small amount of pus. This pus completely blocks off rays passing to the pupil (Bulbus oculi) which therefore remains dark. Other light rays, however, passing into the nasal passage, are reflected from the septum and turbinates into the antrum, producing translucency of the cheek and so giving the erroneous impression that the antrum does not contain pus. On the right is shown the course of rays through the antrum uninterrupted by pus, which thus reach the Bulbus oculi and produce an illumination of the pupil.

In Fig. 2, on the left, is shown the effect of the presence of pus, plus the lack of reflection of light from the nasal passage due to swelling of the lateral nasal wall. Here the absence of translucency of both the cheek and the pupil are noted. On the right, complete filling of the antrum with pus produces a similar result.

Since 1890 the principle of transillumination in rhinologic diagnosis has enjoyed a varying reputation. It has had its staunch supporters and its bitter critics. It has been called a valuable diagnostic aid and a procedure of such uncertain accuracy as to merit its complete abandonment. Series of investigations have been reported tending to show the errors to which transillumination is subject when compared to such methods as puncture or irrigation. Watson-Williams[6] found that it failed to reveal the presence of pus in 20 per cent of his cases of purulent sinusitis and that it gave negative findings in 50 per cent of the cases of nonpurulent sinusitis.

Sheppegrell[7] noted that even after the complete evacuation of pus, the antrum was still dark to transillumination and thus early called attention to the fact that the presence of a purulent or serous fluid is not the only result of an inflammatory process in the sinus, but that changes in the mucosa are present as well and necessarily play an important part in the matter of translucency of the sinus.

In spite, however, of all the criticism directed from time to time against it, transillumination has survived and come down as one of the rhinologist's diagnostic aids, to be regarded by him with

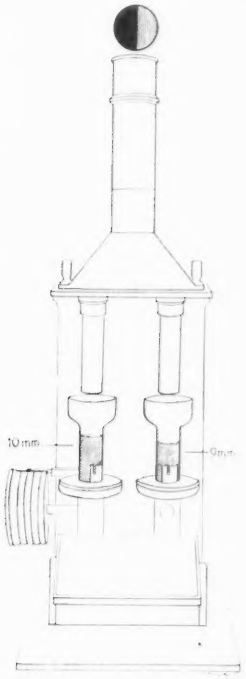

Fig. 3. Diagram to show the use of the Dubosque colorimeter to detect small differences in amounts of a fluid through comparison of light interference.

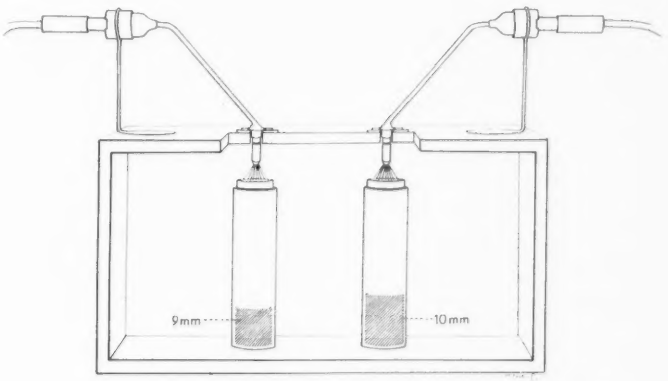

Fig. 4. Diagram of cross section of apparatus for comparison of the translucency of unequal amounts of fluid by the dark adapted eye.

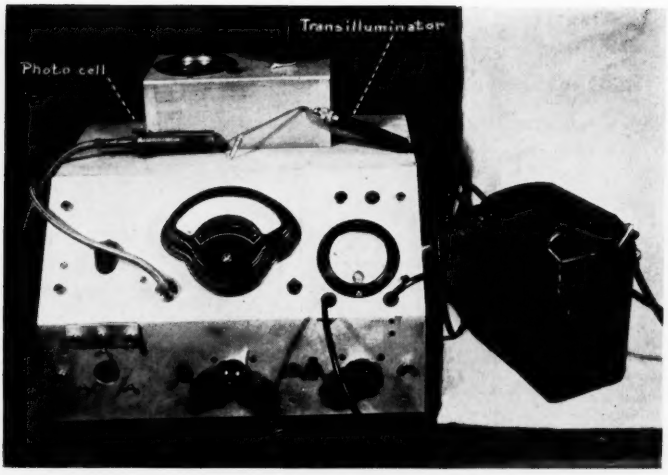

Fig. 5. Photograph of the assembled transilluminometer, with storage battery serving as source of direct current.

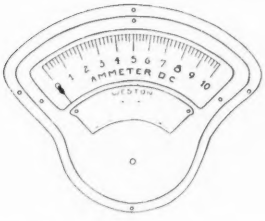

Fig. 6. Facsimile of the dial of the transilluminometer, showing the readings from 0 to 10.

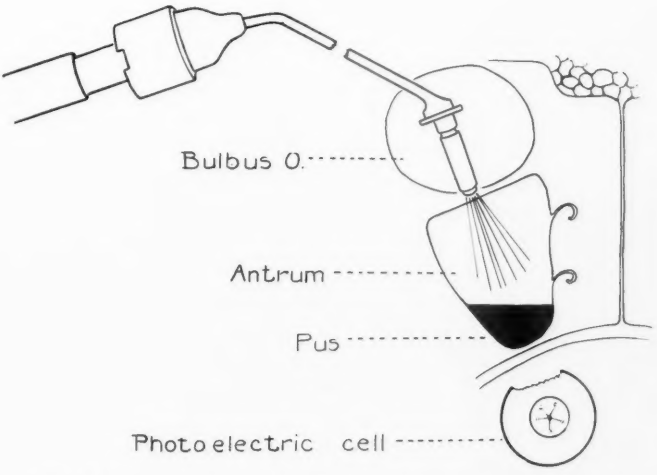

Fig. 7. Schematic drawing to illustrate the manner in which the photocell records the passage of light through the antrum from the transilluminator.

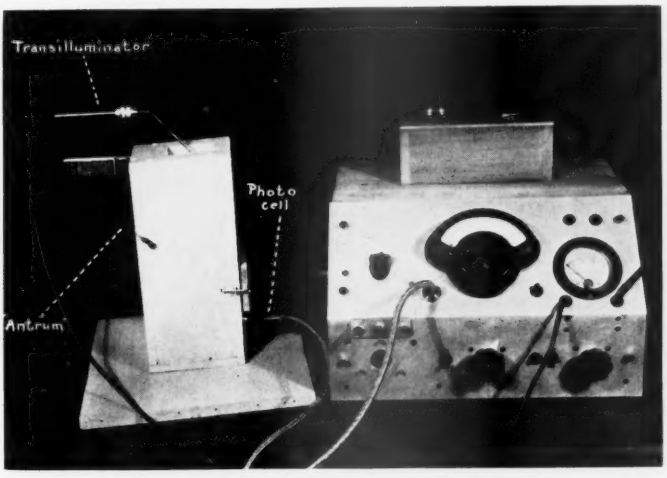

Fig. 8. Photograph of the setup of the transilluminometer and the artificial "antrum."

a degree of enthusiasm proportionate to the amount of help he derives from it. Long ago, in its incipiency, Vohsen[8] claimed that it was at best only one adjunct in making a diagnosis of sinus pathology and such it remains today. Last year Wherry[9] said of it in an article on sinus disease: "I am tempted to voice an opinion that transillumination in maxillary sinus study, even though discredited by some, is of value to the degree of study exercised in its use. To me it is invaluable, especially when a time period for continued observation is possible."

Although it is now some seventy years since the procedure was first introduced, there have been surprisingly few improvements or innovations in the actual technic of transillumination itself. Obviously, the incandescent lamp has made progress since the time when it was necessary alternately to turn on and off the current to prevent burning the patient's mouth, but even the present day forms of apparatus exhibit but little that could truthfully be said to show progress comparable to that observed in so many other types of surgical apparatus.

The only major advance that has been made in all this time is that contributed by Briggs,[10] in 1920, when he called attention to the merits of the so-called orbito-palato-buccal route of transillumination. In this method, the light was inserted not in the mouth, but just above the orbital brim, while the observer noted the degree of translucency seen in the palate on the same side. Briggs claimed for this the advantages that (1) light passed through less extraneous tissue, and (2) it passed through opposite instead of adjacent sides of the sinus, and hence more directly in a straight line. He pointed out in this connection that the average thickness of the orbital wall was between 0.5 and 1.5 mm., while that of the facial wall (traversed in the classical method of observation of the cheek) was from 2 to 5 mm. in thickness. While the latter fact has no bearing if the illumination of the pupil is utilized, it is at once apparent that the method of Briggs eliminates the misleading effect created, as above described, by reflection of light from the nasal chambers. With the light source just above the orbital plate, the rays pass directly through the antrum to be observed directly in the same line through the hard palate. The only valid objection that can be offered against this method is that unless two separate lights are used, each sinus must be tested separately and no opportunity is afforded, as in the older method, for comparing the translucency of the two simultaneously.

Mention has already been made of one fundamental principle involved in the use of transillumination by any method, namely, the

variations in the ability of the human eye to detect differences between unequal degrees of translucency.

It is a well known physiologic fact that the human senses, including the eye, have the ability to distinguish between different amounts of a given sensation. It has been discovered that these different amounts bear a definite relationship to each other. From this fact has been evolved what is known as the Weber-Fechner law. Briefly, this may be stated as follows: If a given sensation is perceived by a sense organ, then the increase in stimulation necessary to be perceived as such bears a definite relationship to the original stimulus producing the first sensation. Thus, if a weight of thirty grams be placed in the hand, then an additional weight of, let us say, three grams must be added before a difference in sensation can be detected. Similarly to a sixty gram weight must be added six grams in order to produce a similar increased sensation, and this ratio continues with each increase in the original stimulus. The particular ratio varies with the particular sense organ involved. For the eye, this ratio has been found within certain limits to be about 1 to 100. In other words, to a light brilliancy of 100 candles, it is necessary to add only 1 candle for the eye to detect the difference. To 200 candles, 2 candles must be added, etc. This ratio, however, holds only for what is known as the light adapted eye, that is, an eye adapted to normal bright light. To the eye adapted to darkness, this acute sensibility of light differences is much diminished to the point that with the dark adapted eye, the ratio must be that of 50 to 100. In other words, to 100 candles must be added a light brilliancy of fifty candles for the dark-adapted eye to perceive the difference. In addition, many other factors relative to the eye itself enter into this ability to detect differences of light, which vary greatly with each individual, according to the state of the eye. All of these factors influence the perception of differences in the translucency of two maxillary antra when compared in this respect. What, therefore, might appear as a definite difference to one examiner might be imperceptible to another, owing to different conditions in respect to his own eye. As noted above, in regard to the Weber-Fechner law, the brighter the lights to be compared, the greater must be the difference between them in order for this difference to be detected. Hence, the less the relative density of the antra, the more difficult would be the comparison between them, except in the event of a considerable difference. Moreover, in transillumination of the sinuses, particularly in that form in which the lamp is held within the mouth, there is nothing to concentrate the two translucent fields to such an extent as to make them easier to

compare. The principle of light comparison in respect to translucency of fluids has long been made use of in biochemical examination in what is known as the Dubosque colorimeter (Fig. 3). Here unequal amounts of a given fluid are transilluminated, the light being collected through two prisms which throw the light from each individual fluid onto a half moon field viewed through an ocular piece similar to that of a microscope. In this manner it is possible for the eye to see the variations in translucency within a single field, side by side, in close approximation, such that by this method it is possible to detect extremely small variations which to the naked eye would not be apparent. Thus, as seen in the diagram in Fig. 3, the comparative difference between a column of a given fluid 9 mm. in depth and one of 10 mm. in depth is at once manifested by differences in the two halves of the circle viewed through the eyepiece. Such an accurate juxtaposition of fields of light, however, is impossible in respect to the clinical use of transillumination of the sinuses. Fig. 4 exemplifies this in an experimental manner. Two columns of serum contained in a dark box and equally transilluminated by two lights shining through them, were viewed in the dark from below through openings in the floor of the apparatus much as the floor of each maxillary antrum would be viewed in clinical transillumination by the Briggs method. It was quite impossible, with the unaided eye, to distinguish any difference between the amount of light passing through the column of 9 mm. of serum and that through the column of 10 mm. of serum. Moreover, since most transillumination is done in the dark, there is usually present the added factor that the eye is at least a partially dark-adapted one and, therefore, is less capable of detecting finer differences in translucency than is the case with the light-adapted eye. While it it true that the eye is not fully adapted to dark in the relatively short time in which transilluminations are carried out clinically, nevertheless, the greatest amount of change toward dark-adaptation occurs in a very short time at the outset and consequently this factor is a distinct one. From these facts it becomes apparent that the accuracy afforded by ocular estimates of differences in translucency as carried out with the present technic leaves much to be desired, and depends greatly on the observer and on the condition under which he is working. In addition, there are bound to be differences in the strength of the transilluminating light used, which may vary on different days or with different bulbs or with variations in the strength of the current used for this purpose. Greatest of all faults, however, is the recognized fact that, whatever the ability of the eye to estimate differences between such translucencies of the antrum, the eye has little or no ability to retain by visual memory a persistent

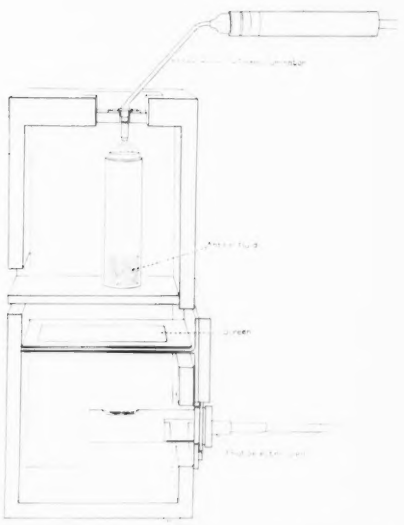

Fig. 9. Cross section of the artificial "antrum" showing the alignment of the transilluminator, "antrum", and photo-cell.

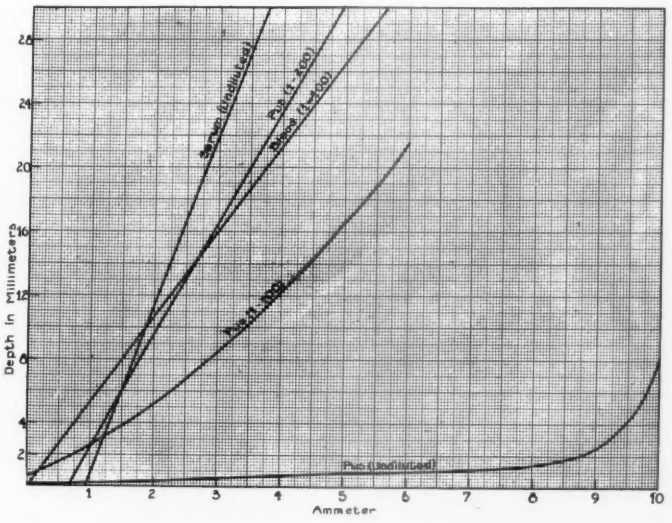

Fig. 10. Graph showing the readings obtained on the ammeter in comparison of different fluids in the artificial "antrum."

picture of this difference. If a given degree of translucency seen on one day is to be compared with a possible change a few days later, there is but small opportunity to recall accurately the original amount of radiance. At best, at the first examination, one antrum may have been recorded as normal or dark or slightly dark or as exhibiting one, two or three degrees of darkness but subsequent findings at a later date have no accurate basis for comparison, except in the presence of the grossest of changes, since there is no metrical record of the first degree of translucency. There would thus seem to be a place for a more accurate measurement of the state of the antrum at the first examination with which to compare possible future changes in some degree comparable to that now available in respect to the ear by means of the audiometer.

The same situation occurs in reverse order in respect to an antrum known to be pathologic in which signs of convalescence are being observed. Here again it is frequently desirable to have a stated density of the antrum recorded with which to compare the progress as the antrum becomes more translucent or less translucent under given forms of treatment. While no such basic normal can be set down as is possible in the case of normal hearing, nevertheless, the condition observed in the absence of symptoms can always be compared with that present under pathologic conditions. Obviously, a maxillary sinus may show a pathologic condition when first observed, and so render impossible any observations of its normal state. Here the opposite side, if uninvolved, will serve as a presumptive normal, though simultaneous bilateral involvement would render this comparison invalid.

As yet I have found recorded but one attempt to achieve any form of permanent record of translucency of the nasal sinuses. In 1933 Wilson[11] described a method of photographing the luminosity produced in the cheeks with a transilluminator held in the mouth. Though the resultant films constitute a permanent record of the sinus condition at the time, and afford an opportunity for comparison with future photographs, the method obviously is as yet too time-consuming and impractical to be of value.

For some time it has seemed to me that some type of what might perhaps be called a transilluminometer might be constructed in a form which would be adaptable to routine clinical use, and which would, by affording a definite metrical reading, supply a need in more accurate work in sinus investigations. The incentive to construct such an apparatus was afforded by experience in photography with the

so-called exposure meter, which depends on the light sensitivity of a photo-electric cell. The apparatus as finally evolved, through the co-operation of workers in the Cruft Laboratory of Experimental Physics at Harvard University consists of a fixed light source, the ordinary Briggs transilluminator, activated either by direct or alternating current, in such a way that the voltage can be accurately controlled and maintained at a constant reading. The other essential part of the apparatus is a photo-electric cell designed to be contained within a metal capsule which can be placed within the mouth and so turned that the open face is directed against the floor of the maxillary antrum. For hygienic purposes this container is covered with a thin casing of cellophane which protects it from moisture and which can be changed with each individual patient. Finally, there is a dial or ammeter on which can be read off the degree of translucency to which the photo-electric cell is subjected. The basic principle involved in the construction of the apparatus is the amplification, by means of a two stage direct current amplifier, of the minute current changes produced in the photo cell. Either a storage battery or alternating current furnishes power for the amplifying tube filaments and the transilluminator. Dry cell batteries furnish potential for the plates of the amplifying tubes and for the photo cell itself.

Fig. 5 shows the assembly of the apparatus, and indicates the relationship of the transilluminator and the photo cell. Controls provide the opportunity for fine adjustments of the dial reading to a baseline[10] of complete opacity. Any light reaching the photo-cell thereafter will be registered on the dial (Fig. 6), which is graduated over a range designed to cover the amount of light which ordinarily passes through the normal human antrum. As more and more light passes through, the hand moves downward from ten to zero, which will be within a range comparable to the greatest degree of translucency which one ordinarily encounters within the antrum, though it is conceivable that an antrum may be so translucent as to be slightly outside this zero range. Thus, when the Briggs transilluminator, with a known brilliancy comparable to that ordinarily employed by most men in transillumination of the antrum, is placed just over the orbital ridge and the photo electric cell, covered with its cellophane, is held lightly within the closed mouth at a specific point determined by a groove which engages the teeth, we then have a system capable of recording with a high degree of accuracy the amount of light passing through that particular antrum (Fig. 7). This will immediately be registered numerically on the dial, thereby affording a definite reading uninfluenced by the human equation. In

the absence of light from the transilluminator, the dial reading will be 10 or extremely close to it, since little or no light affects the cell when held in the mouth. It is, however, so sensitive that when, with the cell in the patient's mouth, an electric light is switched on overhead, there will be a noticeable movement of the dial hand. This, however, can readily be compensated for by one of the adjustments which is capable of bringing the hand directly to 10 as a fixed starting point, so that any change recorded by turning on the Briggs transilluminator will at once be registered as an increased antral translucency. Thus, it is quite evident that whatever the initial variations in reading of an antrum, which may be due to its size or shape or to the thickness of the surrounding bone, provided that there is no known pathology in the antrum, we have here at least a presumptive normal. If subsequent infection takes place in the antrum or pus accumulates within it, or serum is formed within it or if, as a result of a fracture, blood accumulates within it, this change will be registered by a definite change in the dial reading which can be compared with the former figure noted. In the same manner, an antrum known to be infected and full of pus, which is, let us say, translucent only to the point of 9 on the dial at the height of its pathologic state, will be found after irrigation or other therapeutic measures to be gradually returning to a reading of 6, 4 or 2 or whatever the figure may be during the period of convalescence, which change can be recorded at intervals on this basis. Moreover, the use of such an apparatus avoids the necessity for any ocular comparison between various degrees of translucency which may be difficult to detect and eliminates the human variations and error so far as the eye is concerned.

In order that some data might be acquired in the actual workings of such a "transilluminometer," the following investigation was carried out in what might perhaps be termed an artificial "antrum." A completely dark container was built, into which was inserted at the top a Briggs transilluminator. The light shone directly into a glass bottle containing a given amount of one of the common antral fluids, either serum, or pus or blood. Light passing through this fluid then crossed a screen of red paper designed for the purpose of cutting down the amount of light which reached the photo cell which was placed directly underneath it. With this light-tight system illuminated, screening was so adjusted that with the bottle empty, the dial reading was exactly zero. Therefore, the presence of any amount of one or another of the different fluids produced a change in reading toward the completely opaque. This change varied according to the

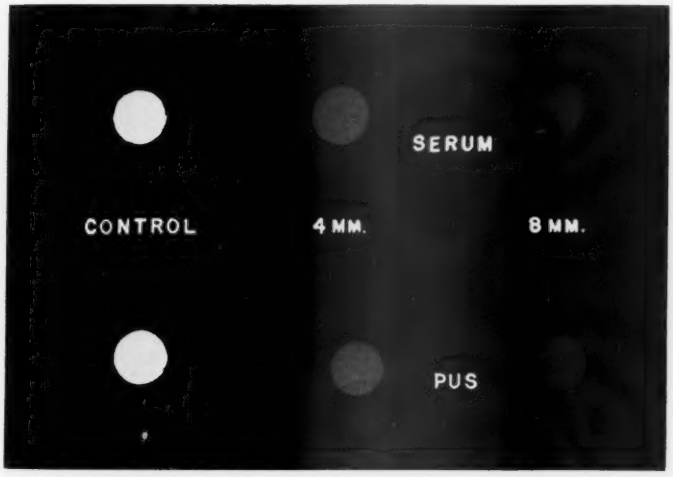

Fig. 11. Photograph of the x-ray film after exposure of unequal amounts of serum and pus.

Fig. 12. Photograph showing the clinical application of the transilluminometer. (The light source should be placed more laterally than is here shown.)

depth of fluid present and also to the character of the fluid itself. In this respect, the system, therefore, represented an antrum into which could be inserted varying amounts of typical fluid. Fig. 8 shows this apparatus as assembled for use. The bottle containing the antral fluid is drawn out on a slide to indicate its position. With the side door closed, the bottle is lowered with its fluid contents and the slide is inserted to the point where the transilluminator, artificial "antrum" and photo electric cell are all in line as shown in the cross section drawing (Fig. 9). With the door shut, the entire system is light-tight, and the only possible variation occurs with a change in the depth or character of the fluid in the artificial antrum. With this apparatus, a study was made of the variations in translucency of three common fluids found in the antra, serum, pus and blood.

Readings of these fluids in different amounts were made on the machine, their average taken and their lines or curves charted. It is a fundamental principle in the translucency of fluids, that in order to compare the translucency of one fluid with that of another, it is first necessary to discover the particular dilution at which an increase in the depth of the fluid will produce a proportionate increase in opacity. When this dilution has been reached, the relationship between the depth of fluid and the opacity, as measured by the photo cell on the dial, can be plotted as a straight line. It was found by repeated experimentation that such straight lines could be obtained by serum alone (undiluted); by pus diluted 1-200 and by blood diluted 1-400. These straight lines, however, did not necessarily coincide, since the translucencies of these three, even with the proper dilution, were all different. When pus was examined at a dilution of 1-200 a curved line was obtained, indicating at once that this dilution was insufficient for comparison with the others. By the use of these graphs (Fig. 10) it was possible to determine the relative translucency of these three particular fluids, using serum as a unit. On this basis, pus undiluted was found to be 260 times as opaque as serum and blood 550 times. In addition, a study was made of the translucency of undiluted pus. It is interesting to note how extremely dense this fluid is and what a small amount overlying, let us say, the floor of the antrum, would be necessary in order to produce a high degree of opacity. It will be seen by Fig. 10 that a depth of only 2 mm. in the artificial antrum produced an opacity registering 9, which is but a short point from 10, or complete opacity. From this, one can derive the interesting clinical observation that an extremely thin layer of pus present on the floor of the antrum is sufficient to produce almost complete opacity from a transillumination standpoint. This is in

direct refutation of one of the objections offered against transillumination, that it would not reveal small amounts of pus. The reason for this objection has already been explained as lying in the reflection of light from the nasal passage, a situation impossible with the method here advocated.

This work also brings out the fact that although serum, such as might be found typically in a cyst, is distinctly less opaque than are purulent fluids of almost any dilution, nevertheless it does offer some interference to the passage of light, especially in large quantities. The clinical observation that an antrum containing a cyst is more translucent than normal is to be explained not on the basis that the cystic fluid is hypertranslucent, but that the bony walls of the sinus have become thinned by the long continued pressure exerted by the cyst. Relatively transparent fluids, such as water and mucus, offer practically no interference to light whatever.

In this connection, some attention was devoted to a comparison between the transilluminometer and the x-ray with regard to their ability to detect differences between different fluids and unequal amounts of these. Fig. 11 is an x-ray film exposed to typical pus and serum. To the left are control exposures containing no fluids. In the center was placed a container with 4 millimeters' depth of pus and 4 millimeters' depth of serum. To the right were placed containers with 8 millimeters' depth of pus and serum respectively. A single exposure was given to all four. It is plainly evident that the x-ray can detect a difference, though not a marked one, between 4 millimeters of pus and 8 millimeters of it, likewise between 4 and 8 millimeters of serum. There is, however, but little detectable difference between these same amounts of pus and of serum, a situation quite different from that recorded by the transilluminometer. In other words, the x-ray is less sensitive to the density of the fluids than to their amounts. Density as well as amount, however, has a profound effect on the translucency of the fluids.

Fig. 12 shows the actual apparatus in use on a patient. The transilluminator itself is used exactly as in the method advocated by Briggs. The photo electric cell container is held within the closed mouth, its opening rotated slightly to receive the maximum rays coming through the antrum. The reading on the dial is then noted and recorded. It is quite unnecessary to darken the room, the only adjustment required being the setting of the dial at 10 after the photo cell is placed in the mouth, before placement of the lamp over the middle of the intra-orbital ridge. The apparatus, like many of

its sort, is a first attempt in an effort to solve a problem. It has its obvious defects. It is not, as yet, as portable as one would like, and the container for the photo electric cell is not as small and delicate as it may be possible to obtain with future development. The apparatus was no sooner completed than the physicist responsible for its technical features saw obvious ways in which its construction could be improved. It is ever thus with any mechanical contrivance. The one outstanding factor which has caused some difficulty is the variation in reading obtained by changes in position of the transilluminator. When this is pressed deeply into the orbit, a different reading is obtained than when it is pressed lightly. Also there is some variation according to whether the light is held toward the lateral aspect of the orbital rim or toward the medial aspect. These variations are all equally present when the transilluminator is used in the conventional manner, but the variations in light not detectable by the eye are at once registered on the photo electric cell. However, it is quite possible to develop a technic such that a standard position of the light and a constant degree of pressure are exercised, in which case, without variation in the antrum itself, a constant reading will be obtained. The very sensitivity of the apparatus thus introduces at first some difficulty but will subsequently prove to be its major feature. A certain degree of practice, however, is necessary in order to achieve this sense of touch. It should be added in connection with Fig. 4, which shows the inability of the eye to detect differences between 9 mm. and 10 mm. of serum, that this difference in fluid depth is at once detectable by differences in the reading on the photo cell dial. The apparatus is not offered as the acme of perfection or the final evolution along this line. It is felt that it approaches a distinct advance toward an objective and provides an additional accuracy not heretofore obtainable in ordinary transillumination. It will not distinguish between fluid and thickened membranes nor determine definitely whether a small amount of pus or a large amount of serum is present. It will not serve for transillumination of the frontal sinus, which must be carried out in the customary manner, in full recognition that the variable shape and size of the frontal sinuses make the procedure quite unreliable. It does not in any way supplant the x-ray as a valuable diagnostic adjunct. It would seem to be of use in avoiding the necessity of repeated x-rays to check the clinical progress of a given case. It aims to furnish a method of repeated measurable observations in a clinical situation which has heretofore been quite unsatisfactory, and it is hoped that with future perfection of the apparatus it may become of use to others.

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XII.

THE FUNDAMENTALS OF ALLERGIC RHINITIS WITH PARTICULAR REFERENCE TO IONIZATION (IONTOPHORESIS).*

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In almost every discussion of nasal sinusitis in children it has been said that a most important part of the treatment of the condition is to eliminate allergic rhinitis. This has been a very difficult problem. In many instances it has not been solved.

Allergic rhinitis is a most important etiologic factor in nasal sinusitis, and if it is not controlled the infection of the sinuses will persist. Many children with chronic purulent nasal sinusitis have a definite history of attacks of acute allergic rhinitis early in infancy. If an infant is sensitive to milk and develops an allergic rhinitis which lasts for months and then spontaneously disappears, the nasal mucosa does not return to normal. Most of the serum-logged intumescent nasal membranes, whether allergic or non-allergic that we see later in life have their beginning in infancy. This thickened membrane predisposes to blocking of the ostia of nasal sinuses with the production of stasis. Stagnant fluid in nasal sinuses always results in infection.

The most important thing in the treatment of allergic rhinitis is a proper diagnosis. To make such a diagnosis it is necessary to study the patient for two hours a day for at least ten days. Often more time is needed. Most of the work is done by expert technicians so that a minimum amount of time is required of the physician. With all the information secured it is not difficult to outline a course of treatment and make a reliable prognosis.

Many poor results are due to the fact that this thorough study is not made or because the physician uses only one type of therapy. It is necessary, after the diagnosis has been made, to select from a

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group of therapeutic measures those that are applicable to the particular case. Certainly neither ionization nor desensitization is indicated in all cases. If this plan is carried out one will have a large group of satisfied patients without doing anything to the nose that will produce a deleterious result.

The patients with allergic rhinitis that we see in the late fall, winter and early spring are mostly of the perennial type. We insist that these people give us the necessary time to study the case. We wished to study the absorption of allergens by the nasal mucosa before and after ionization. As a direct result of this comprehensive study of our cases in the three months we have not found a case where we were justified in recommending ionization.

While these patients have had good subjective results their noses have not returned to normal. We make a biopsy of the membrane before and after the treatment. Patients with perennial allergic rhinitis that have suffered from sneezing, nasal stoppage, nasal discharge and repeated colds for years, may get an excellent subjective result from the avoidance of allergens. The mucous membrane of the nose does not return to normal. The turbinates are decreased in size but the membrane remains pale in color and somewhat boggy. The biopsy shows some hyperplasia of epithelium, some thickening of the tunica propria with definite edema.

In the patient we studied most carefully the eosinophils which were so abundant in the tissue before treatment, disappeared from the tissue after treatment. This observation needs confirmation. In the one case of allergic rhinitis studied carefully before and after ionization the eosinophils in the tissue disappeared after the ionization. This must have been a coincidence, because in all other post-ionization biopsies of cases of allergic rhinitis, the eosinophils have remained in the tissues.

The following is an outline of our method of studying suspected cases of allergic rhinitis:

I. History
II. Examination of the ear, nose and throat
III. Nasal smears
IV. Blood count
V. X-ray examination
VI. Bacteriological examination
VII. Dietary deficiencies

VIII. Endocrine disturbances
IX. Skin tests
X. Intramucosal tests
XI. Passive transfer
XII. Test for bacterial allergy
XIII. Study of leucopenic index
XIV. Result of avoiding certain allergens

The history is a most important thing and yet in evaluating the history one must use common sense. The fact that a patient was sensitive to milk some years ago does not mean that he is sensitive today. The fact that a patient has an outstanding gastro-intestinal allergy does not mean that a co-existing nasal pathology is necessarily allergic. In known cases of gastro-intestinal allergy we have secured the most excellent results in marked nasal blockage of seven or eight years' duration by the use of diet, exercise, vaccine therapy and by making the nose normal. I see no reason for suspecting that the nasal condition was allergic.

As emphasized by Rachemann, in all cases of acute allergy the dates on which the patient had the acute upsets is most important. A study of the contacts on these dates may solve the problem.

In every case of allergic rhinitis the nose and throat must be studied, because the treatment of almost every case of allergic rhinitis consists of the treatment of the pathology in the nose plus a proper immunologic therapy. During the last year we have seen only one case of allergic rhinitis with a nose that was clinically normal. This patient had had one mild attack of ragweed hay fever and a second season with the symptoms more marked. She reported no trouble except during the ragweed pollen season. Repeated attacks of her allergic rhinitis may leave the nose in such a condition that during the winter she will suffer from nasal stoppage, particularly if she is exposed to drafts, if her feet come in contact with the cold floor or if she has a mild upper respiratory upset.

A person who suffers from ragweed hay fever may have, during the winter, symptoms of allergic rhinitis. These can be controlled by nasal procedures, leaving the patient still subject to the attacks of seasonal allergic rhinitis. The more normal the nose the less difficulty will be experienced with a given attack of acute allergic rhinitis. An exception to this statement is the presence of atrophic rhinitis.

Patients with atrophic rhinitis do suffer from hay fever, but the nasal mucosa undergoes only the most moderate swelling and

there is no nasal blockage. This suggests how all procedures which splint the mucous membrane of the nose do favorably influence the attacks of allergic rhinitis. Certain things can be done in the nose which favorably influence allergic rhinitis without splinting the mucous membrane. If adenoids are present and they are removed, or if there is a deflected septum or exostoses causing nasal blockage, an improvement may be secured without deleteriously influencing the condition of the mucous membrane of the nose.

On one day I have seen three children with allergic rhinitis, one treated by the removal of adenoids, a second by ionization and a third by the avoidance of allergens, all three with equally good results. We have a patient with perennial allergic rhinitis whose diagnosis was made by a thorough examination, including a biopsy from the turbinates. The biopsy showed a typical allergic picture. Nevertheless the patient secured complete subjective relief with objective shrinking of the mucous membrane following a submucous operation. Immediately the question is raised, "How long will this patient be benefited?" The answer is not definite, but the question might be answered in this way: "How long will a patient be benefited by the avoidance of allergens, the use of ionization or any other treatment for allergic rhinitis?" The truth of the matter is that the fundamental allergic status of the individual persists regardless of the procedure used to secure the desired result. If desensitization is used, we do not desensitize the patient; we simply get the patient in a refractory state. So I feel that the examination of the nose and throat is an essential procedure in every case of allergic rhinitis. Further, I feel that every case of allergic rhinitis must be taken care of by the rhinologist. If the rhinologist prefers to have the immunologic study made and the treatment given by another person, I see no objection to this procedure, providing the contact is sufficiently close between the two men concerned to give the patient the very best service. It is certain that the patient is going to get the greatest relief by having a complete immunologic study and a rhinologic examination made.

In every suspected case of allergic rhinitis smears prepared from the nasal discharge should be studied. The finding of an excessive number of eosinophils in the nasal smear does not make the diagnosis of allergic rhinitis. We all see patients with parasitic disease, with digitalis poisoning or other similar conditions that have an enormous number of eosinophils in the nasal discharge. These patients, however, do not have edematous nasal mucous membranes. So far, every patient that I have seen with vasomotor rhinitis plus a large number of

eosinophils in the nasal discharge has proven to be a case of allergic rhinitis. We may find an exception to this statement in the near future.

In order to determine the relationship between eosinophils in the nasal discharge and allergic rhinitis, Miss Zelma Alvis, in our service, studied a group of sixty-nine persons with no history of or clinical evidence of allergic rhinitis from the standpoint of eosinophils in the nasal discharge. The following table illustrates her findings.

Allergic study of six cases from a group of sixty-nine persons without clinical allergic rhinitis that show an increased number of eosinophils in the nasal smears (Alvis):

Patient	Nasal smears eosinophils	Blood eosinophils	Skin tests
C. B.	4 plus	9%	Plus-minus: dust
F. D.	3 plus	5%	Plus-minus: dust, orange
A. A.	3 plus	2%	3 plus: ragweed
J. B.	2 plus	8%	———
L. O.	2 plus	4%	———
W. Y.	2 plus	5%	Plus-minus: wool, egg orange

The nasal examination was quite negative for allergic rhinitis except in case of L. O., where there was a suspicious edema of the posterior ends of the middle turbinates.

Four plus equals very many eosinophils.

None of these patients gave a history of allergic rhinitis. On clinical examination none of them except the fifth showed any suspicion of allergic rhinitis. In other words, there were five patients with a great increase in the eosinophils in the nasal discharge who were not suffering from nasal trouble and in whom there was certainly no reason for treating the nose. It is interesting to note that two of the six patients had a significant increase in the eosinophils in the blood. The only conclusion that we wish to draw from this study is that an increase in the eosinophils in the nasal discharge is not in itself sufficient to make a diagnosis of allergic rhinitis.

A blood count should be made with particular reference to the number of eosinophils present. One should know the thickness of the lining of the nasal sinuses, hence an x-ray examination is important. It is axiomatic that a bacteriologic examination, looking for anaerobes as well as for aerobes should be made.

Dietary deficiencies are most important. In our service we were without an efficient nutritional aid for a period of five months. We are all of the opinion that our patients with chronic infections who were being treated by the use of vaccine did not secure the good results which were secured when we had this aid. It is certain that the correction of dietary deficiencies does help in the management of allergic rhinitis. Certainly if we take out from the diet a food, because we suspect the patient is sensitive to that food, it must be replaced by some other food that is equally nutritious. We consider the dietary part of the work so important that we have hastened to again add to our service a very expert dietitian.

Malnutrition by decreasing the resistance of the cells of the mucous membrane to the entrance of allergens into the tissues and trauma of the epithelial cells are important factors in the sensitization of the organism. A well nourished mucous membrane does resist the entrance of pollens into the organism better than a poorly nourished one. A break in the protective epithelium permits the easy entrance of allergens. Six years ago, when I was holding a clinic, a case was brought to my attention of asthma following tonsillectomy. For years we have known that operations on the nose rarely cause asthma. It may follow cautery of the nose. Now we are hearing of many cases following ionization.

In each instance there was a break in the protective epithelium which permitted the allergens to enter the tissues and produce sensitization. It is interesting to note that in at least one case the patient suffers from allergic upsets only when he is in the community where he was operated upon. This has led us to advise in some cases that these patients change their environment.

Endocrine disturbances should be sought for and corrected. During the coming year we will make a study of the relationship between the lesions of the ductless glands and allergic conditions. We are inclined to consider this a very fertile field.

We make skin tests in every case. The reason for testing the skin is to get a more or less definite conception of the allergic condition of the patient. We realize that skin tests may give us more an idea of the past allergic history of the patient than the present allergic status.

Reagins are found in all the tissues of the body except the cerebro-spinal fluid and the aqueous humor of the eye. More of these reagins are produced in the skin than elsewhere. As soon as the acute allergic upset is past the elimination of these antibodies begins. They

are eliminated first from the blood and apparently last from the skin. Because of this the patient may remain skin sensitive for many years.

So many cases of allergic rhinitis were relieved by ionization and other procedures that we were inclined to question the diagnosis. In many of these cases the diagnosis was made because of nasal trouble, with some increase in eosinophils in the nasal discharge and positive skin tests. Because of this we were inclined to doubt the diagnostic significance of positive skin tests plus some increase of eosinophils in the nasal discharge as being definitely indicative of allergic rhinitis. Consequently we made a study of the eosinophils in a group of individuals as mentioned above. Skin tests were also made on sixty-nine individuals with no history of or clinical evidence of allergic rhinitis. The following table gives us the results of this study by Miss Alvis:

Number tested		69
Number giving definite positive skin reactions with pseudopodia		11
Allergens giving positive skin reactions:		
Ragweed	Orris root	Chocolate
Wool	Mixed feathers	Orange

An examination of the noses of the eleven patients giving positive skin reactions was made in each case by some member of our staff. They showed no evidence of allergic rhinitis at the time the skin tests were made. Neither was there any history of this condition.

Because of our lack of confidence in the skin tests as indicative of allergic rhinitis, Mr. Linton a year ago began making intramucosal tests.[1] A comparison of skin and intramucosal tests by Mr. Linton will soon be published. This proves conclusively that skin sensitivity is not an indication of the sensitivity of the nasal mucosa. The following case illustrates this:

W. B.—Case of allergic rhinitis. Gave nine plus-minus skin reactions. Only two of the nine allergens gave positive intramucosal tests. Feathers gave negative skin reactions, but a very strong intramucosal reaction. Avoidance of feathers gave complete relief.

We depend on the intramucosal test more than anything else in determining the allergic status of the nasal mucosa. It is a very exact procedure. If one avoids the use of sera in making these tests and if in using the pollens and foods the allergen is sufficiently diluted, there is little or no danger in making the tests. We have made many hundreds of tests and have had a severe reaction in only one case. It is interesting to note how many patients with allergic manifestations, such as asthmatic attacks, headaches, blocking of the ears, etc., get

similar reactions when given intramucosal tests. These findings are important therapeutic leads.

The best way to get a definite knowledge of the systemic allergic status of an individual is to make a passive transfer test. This is an easy thing to do but is very difficult to interpret. One of the difficulties is that by using individuals, all apparently equally nonallergic, we get decidedly different reactions. That is, with the same blood we may get a plus 1 or a plus 2 reaction with different individuals who are apparently the same as far as their allergic status is concerned. I think this is one of the reasons why passive transfers made before and after a certain treatment often gives varying results when, as a matter of fact, there is no change in the allergic status of an individual.

Tests should be made for bacterial allergy. This requires from ten to fourteen days. Before the test can be made the vaccine must be prepared. In bacterial allergy, using the vaccine for testing, we get the same skin reactions as with other allergens. When the individual has a severe systemic reaction to exceedingly small doses of vaccine, he has bacterial allergy. The term "bacterial allergy" should not be used unless we are going to speak of ragweed allergy, milk allergy, etc., because there is no difference between bacterial allergy and any other kind of allergy. It produces the same changes in the nose; that is, vasomotor rhinitis with an increase in the eosinophils in the tissue and in the nasal discharge with marked edema and thickening of the tunica propria.

Multiple sensitivity is the rule in allergic rhinitis. Consequently if the nose is found sensitive to one allergen, sensitivity to others should be sought. Sensitivity to foods should be looked for, particularly in ragweed allergy.

Not only do we have a multiple sensitivity, the result of sensitization by contact with various allergens, but a person who has been sensitized to one allergen may react to others because of the similarity of the molecules; that is, other substances besides the one that has produced the sensitivity may cause a reaction. Fortunately in the same way desensitization of an individual to one substance may make him refractory to other substances.

As a part of our diagnostic procedure we furnish the patient with a table which has been worked out by Dr. Proetz. In this table the patient notes the various allergic difficulties that he has every day, the time of the reaction and in the same table he sets down his contacts on that day.

The leucopenic index, as worked out by Drs. Gay and Ritter, has proved to be of great value as an exact procedure in the study of food allergy. We have studied the leucopenic index in many cases of known sensitivity to foods. In each instance a typical decrease in the blood count has been found.

In a few cases of perennial allergy the leucopenic index, together with the subjective reaction at the time the food was given, has helped more than anything else in the diagnosis of the systemic allergic condition which accompanied an allergic rhinitis.

There is a nonallergic vasomotor rhinitis. As to whether or not all of these patients have at some time suffered from allergic rhinitis, I would not care to say. I will say that I have seen a case of chronic nonallergic vasomotor rhinitis where the swollen tissue completely blocked the nose and where I was positive at the first examination that it was a case of allergic rhinitis. This patient suffered from malnutrition. On a proper diet, week after week, I saw the mucous membrane of the nose shrink. After a year of study we have been unable to find a single indication that this person is now or ever has been allergic. All skin and intramucosal tests were negative.

Ionization has been recommended for allergic rhinitis. Dr. Wenner[2] has shown this to be true iontophoresis, the zinc entering the tissue.

Ionization acts by thinning and splinting the mucous membrane. It interferes with the absorption of pollen through the mucous membrane of the nose. The columnar ciliated epithelium is destroyed and in its place we find a hyperplastic and metaplastic epithelial lining. The surface cells have a tendency to become squamous. There is a tendency for this metaplasia to disappear and ciliated cells to be again produced. In the human nose I have never seen a complete return of ciliated cells; metaplasia persists in areas. A definite result of ionization of the human nose is an absence of or a great decrease in the number of goblet cells. This has been present in every case studied.

I have seen a biopsy from a human nose ionized three months before with areas of perfectly well developed ciliated cells. The clinical examination has shown the mucous membrane to be so dry and the mucus so thick that it would be impossible for the cilia to carry out their function. It has not been demonstrated that the activity of the new cilia is normal. We have tried to compare the activity of cilia in un-ionized and ionized noses. As yet we have made no progress with this investigation.

A common result of ionization, which lasts at least two years, is a thickened and more dense basement membrane. There is fibrosis of the tunica propria which in the human nose I have never seen disappear. Two years after the ionization it often looks like scar tissue. I have seen the epithelium in shallow folds. Dr. L. C. Montgomery has suggested that this may be a reflection of the fibrosis. I have not noted any constriction of ducts or acini of glands by this fibrosis. In mucous membranes, one to two years after ionization, we almost always find inflammatory changes adjacent to the acini of the glands.

The mucous glands of the membrane undergo hyperplastic changes. In most of the glands in a year or so this disappears. It has not been proven that the function of these glands is normal. Some of the glands are atrophied.

There is a more or less complete paralysis of the parasympathetic nervous mechanism of the nose. This is probably due to the destruction of the end organs of the parasympathetic nerves. Dr. Wenner and Mrs. Alexander will soon publish a study of the action on the parasympathetic nervous system. This interference with the parasympathetic system is either incomplete or temporary, as is evidenced by the positive reaction of ionized noses to intramucosal injections of allergens as reported by Mr. Linton.[4] The reaction of the nasal mucosa to the intramucosal injections shows that the sensitivity of the membrane is not altered. A person sensitive to ragweed pollen after ionization usually gives no reaction if the pollen is applied on cotton to the mucous membrane of the nose. The reaction is always present if the pollen is injected into or underneath the epithelium of the turbinate.

Next year we will study the relative absorption of allergens through untreated and treated mucous membranes. One patient who had one side of the nose ionized has already been studied. The method of determining the absorption of allergens through the mucous membrane of the nose, described by Sulzberger and Vaughn,[3] was used. The patient was nonallergic to ragweed. Blood containing reagins to ragweed was injected into the skin of one arm. A similar injection of blood without reagins was injected into the other arm. After twenty-four hours ragweed pollen was placed in the un-ionized side of the nose. In fifteen minutes there was a reaction on the arm at the site of the injection of blood containing reagins. The other arm did not react. This was repeated on the ionized side of the nose. There was no reaction in either arm.

Dr. Wenner[2] demonstrated in the web of a frog and in the ear of the rabbit the slowing of the blood stream in ionization. This he

believes is the cause of the superficial necrosis. Ionization does not affect the lining of the nasal sinuses. Examination of the lining of the maxillary sinuses of guinea pigs eight days after ionization showed no change. The lining of an ethmoid cell removed from a human subject three months after ionization was not affected by the ionization.

We have been unable to study the cartilage of the bone in the human being. In animals after ionization there has often been a death of the cartilage cells of the septum on the side ionized. This may have been due to the electrodes being too intimately in contact with the septum.

The final results of the ionization of the human nose, as I have observed them, are: first, fibrosis with thinning of the tunica propria; second, hyperplasia of the epithelium with some metaplasia; third, a partial destruction of glandular function, and fourth, a decided decrease in the goblet cells. There is often a thickening of the vessel walls, particularly the capillaries. Some of the acini of glands appear to be completely obliterated by the cellular hyperplasia. In others the secretory cells are atrophied. The basement membrane does undergo a marked thickening and it is more dense. These changes result in an interference with the secretory function of the nasal membrane and the membrane does not swell as easily as before. With the change in the epithelial lining and the interference with the secretion of nasal discharge, together with the formation of a thickened and more dense basement membrane, it is easy to understand why the absorption of pollen is interfered with.

While this contribution was being prepared a person who was ionized two years ago came to the clinic complaining of postnasal discharge. The history suggested that she was ionized because of perennial allergy. Since her ionization she has been free of a very troublesome nasal obstruction. At this time the patient presents a mild rhinitis sicca. The middle turbinates were covered with thin crusts. With the removal of the crusts, superficial excoriations were found. The mucous membrane was paler and dryer than normal; there was no nasal obstruction. Nasal smears showed an increase of eosinophils and many polymorphonuclear neutrophiles. The blood eosinophilia was 8 per cent.

A biopsy was taken from the middle third of the inferior turbinate. There was a hyperplasia of epithelium, some areas showed ciliated cells, others were covered with squamous cells; there were no goblet cells. The basement membrane was much thickened and more

dense than normal. The tunica propria was thin and fibrous. The fibrous tissue looked like scar tissue. There was a scattering of eosinophiles in the tissue; they were slightly increased in number. Most of the glands appeared to be functioning; many were permanently atrophied. Around the acini were distinct inflammatory changes. The walls of the capillaries were thickened. This was not true of the larger vessels. This is the only biopsy of human ionized turbinate that we have studied where there was no edema. Intramucosal tests produced subjective reactions, headache, feeling of malaise but no nasal stoppage; that is, the turbinates did not touch the septum.

Certainly with these changes in the membrane, one would hesitate to do repeated ionizations. True atrophic rhinitis cannot result from ionization; rhinitis sicca is often present after the ionization. It has lasted at least two years. The more allergic the individual, the less persistent is the dryness of the membrane. I mean to say that both the splinting and the dryness persist longer in cases of non-allergic vasomotor rhinitis. A severe attack of hay fever in an ionized nose is almost certain to leave behind evidences of vasomotor rhinitis. With repeated attacks of acute allergic rhinitis the pale color of the membrane decreases, nasal obstruction with sneezing reappears.

The only improvement which one gets in a case of ragweed hay fever where the patient is exceedingly sensitive to ragweed pollen, as evidenced by plus 4 reagins in the blood stream, is a slight decrease in the nasal obstruction during the attack of hay fever. In the very sensitive ragweed patient apparently the small amount of pollen absorbed through the ionized membrane causes a reaction. We have taken a patient who had a mild sensitivity to wheat flour, as evidenced by a positive skin and a positive intramucosal reaction, but with no reagins to wheat flour in the blood stream. This patient was very sensitive to ragweed pollen and had plus 4 reagins for ragweed in the blood stream. She was ionized in midwinter and the chronic vasomotor rhinitis from which she suffered disappeared. She was completely relieved of the swelling and sneezing when exposed to flour particles. An intramucosal injection of ragweed pollen produced an attack of acute rhinitis that could only be controlled by hypodermic injections of adrenalin. It was easy to predict what would happen to this patient during the hay fever season. At that time she received little or no relief from the ionization.

Ionization does improve most cases of allergic rhinitis. The less the sensitivity to allergens, the greater the relief. Ionization does produce deleterious results in the mucous membrane of the nose which last at least two years. Many changes are permanent.

There is no excuse for using ionization if procedures that do not produce deleterious results will give a satisfactory result. Ionization should not be used unless the benefits derived are greater than the deleterious results produced.

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XIII.

HOW DOES THE ORGAN OF CORTI DISTINGUISH PITCH?*

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In the past five years there has been an increased interest in the physiology of hearing. This new impetus was given in 1930 by Wever and Bray[21 22] of Princeton, when they reported the phenomenon now known by their name. Work has been done at various universities since then and a great many reports have been made. Many of these reports have been in direct contradiction to one another, resulting in confusion to the interested otologist. In this presentation I shall endeavor to correlate the results of our investigations at the Harvard Medical School[5 6 7 8 16 17 18 20] with those of the neuro-anatomist and the clinical findings of the otologist.

A fundamental point to be settled is the question as to what vibrates in the membranous cochlea when a tone reaches it.[9 17 20] In one of our experiments we sent to the animal's ear a tone of 400 cycles per second (d.v.) at an intensity of 125 decibels (db.) above threshold. This is a loud tone. The animal's hearing before this happened was normal as judged by its electrical response. After the exposure to the tone there was a loss of sensitivity from 50 to 70 decibels (db.) throughout the hearing range. On sectioning the cochlea it was found that the outer portion of the organ of Corti had been thrown off the basilar membrane in a turn and a half and the rest of the organ of Corti was damaged throughout the cochlea. The internal hair cells remained in their normal position. The tectorial membrane was intact and in its normal relationship to surrounding structures. Reissner's membrane was normal. The basilar membrane showed no evidence of damage and the basilar artery was not ruptured. But the damage that occurred was the dislodging of that part of the organ of Corti which overlaid the most movable portion of the basilar membrane. Here was direct evidence that the basilar membrane does vibrate and that the organ of Corti is affected by the

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movement of the basilar membrane. This experiment was performed three times with similar results. (Figs. 1 and 2.)

The neuro-anatomy of the organ of Corti and the primary cochlear nuclei of the medulla have been brilliantly described by Lorente de No.[13 14 15] I would like to present a summary of his findings, as they have an important bearing on our subject. Lorente de No describes the nerve supply of the organ of Corti as being rather complex. He demonstrates that the nerve fibers supplying the internal hair cells connect with one or two hair cells, at the most. These are called the radial fibers. The external hair cells are supplied by the external spiral fibers which run below the hair cells, and a single ganglion cell will connect with a large number of external hair cells, for a third of a turn or more. The internal spiral fibers are a group of fibers running below the internal hair cells and establish connections not only with internal hair cells but also with external hair cells. The diagram taken from his work clearly shows this nerve supply. (Fig. 3.)

Lorente de No then postulates that with the resonance theory of hearing there must be a projection of the ganglion of Corti (spiral ganglion) in the primary cochlear nuclei. If the telephone theory is correct, then there is no need of any such projection, since the interpretation and analysis of sound would be done in the cerebral cortex. For the resonance theory there must be specific endings in the primary cochlear nuclei for different parts of the basilar membrane. As he rightly points out, one cannot assume resonance for the cochlear mechanism without specific fibers in the hearing tracts. He then presents the evidence of definite projection in the primary cochlear nuclei. The cells of the ganglion of Corti are distributed along the cochlea parallel to the organ of Corti. From the ganglion cells there are given off two nerve processes, one going to the cells in the organ of Corti, the other forming the cochlear nerve going to the medulla. The nerve fibers of the cochlear nerve are twisted as a result of the embryonic development of the cochlea. The ganglion cells are present before the organ of Corti is developed, and the cochlear nerve has already made its connection with the primary nuclei of the medulla. Then with the spiral growth of the organ of Corti there is also a twisting of the spiral ganglia (ganglia of Corti), and, as a result, a definite twisting of the fibers in the cochlear nerve external to the medulla. But the nerve endings of the cochlear nerve in the primary nuclei do not twist, and there is a spatial representation of the organ of Corti present, shaped like a boomerang, and this is a projection of the ganglion of Corti as it would be uncoiled. S.

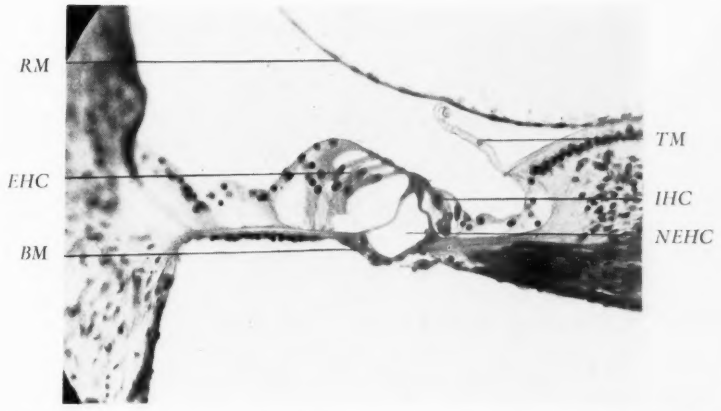

Fig. 1. Normal organ of Corti of guinea pig, 300 x, showing normal relationship and appearance of the external and internal hair cells. The nerve fibres going through the tunnel of Corti to the external hair cells. The normal appearance of tectorial membrane and Reissner's membrane. *BM*, basilar membrane; *EHC*, external hair cell; *IHC*, internal hair cell; *NEHC*, nerve to external hair cell; *RM*, Reissner's membrane; *TM*, tectorial membrane.

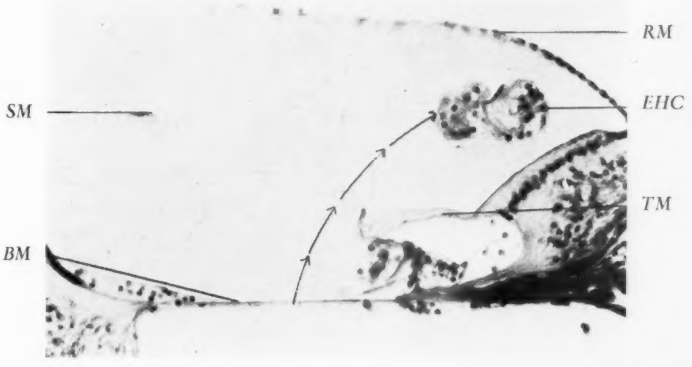

Fig. 2. Organ of Corti of guinea pig, 300 x. Animal had been exposed to tone of 125 decibels (db.) in intensity. Note that the external portion of the organ of Corti has been thrown off the basilar membrane and is lying free in the scala media. The tectorial membrane is still in normal relationship to the organ of Corti. *BM*, basilar membrane; *EHC*, external hair cell; *RM*, Reissner's membrane; *SM*, scala media; *TM*, tectorial membrane.

Guild[3 10 11] and his co-workers have reported that there are more ganglia cells in the central region of the cochlea than at the apex or base. Lorente de No shows that this is true in the projection present in the primary nucleus. Thus the area of the greatest discrimination of sounds, from the neurologic point of view, is the central portion of the cochlea, as this part receives the greatest nerve supply. Figures from his (L. de N.) article on the anatomy of the eighth nerve show this projection very clearly. (Fig. 4.)

S. Poljack, in his monograph, "The Main Afferent Fibre Systems of the Cerebral Cortex in Primates," also comes to the conclusion that there is a definite projection of the auditory fibers in the cortex at the region of the Sylvian sulcus. The findings of the neuro-anatomist are definitely in favor of the resonance theory of hearing.

From the physiological experiments carried out in the Department of Physiology at the Harvard Medical School we have also obtained evidence of the localization of pitch perception on the basilar membrane.[7 20]. The experiments were performed on guinea pigs because the cochleas of these animals are easily exposed and operated on. The guinea pig has four and a half turns in the cochlea. By drilling holes in the cochlea at different places we were able to destroy or damage the organ of Corti at various turns. The cochlea was tested immediately after damage for its greatest rise in threshold to stimulation. It is important in this type of experiment to work with the smallest intensities, as we have found that an increase of sound to 20 decibels (db.) above threshold stimulates such a large portion of the basilar membrane that no clear evidence of localization can be obtained. The cochlea is then examined microscopically in serial sections and the lesion found accurately located. The damaged region and the definite tone loss found were plotted, using the frequency of tone as the base line. It soon became evident that octaves in different parts of the hearing range do not occupy same distances along the basilar membrane. Tones below 500 were crowded together toward the apex, and the tones above 8,000 were crowded toward the base. The range beteen 500 and 8,000 occupies two-thirds of the cochlea.

Shower and Biddulph[19] have presented curves on differential pitch sensitivity of the human ear. They have shown that the human ear is able to distinguish about 1,300 pitches between the lowest and highest audible tones. By integrating the data they presented, a curve was obtained which practically coincides with the curve plotted by the method just described. Culler of Illinois,[4] in experiments conducted by an entirely different method, has presented evidence in agreement with our findings and he plots the localization of pitch

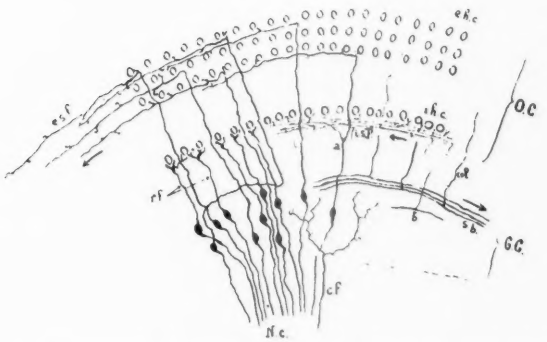

Fig. 3. Reproduction by permission of R. Lorente de No from the article "The Reflex Contractions of the Muscles of the Middle Ear as a Hearing Test in Experimental Animals." Published in the Trans. Amer. Laryngol., Rhinol. & Otol. Soc., 1933, 26-42. This demonstrates the nerve supply of the external and internal hair cells in the organ of Corti. *i.b.c.*, internal hair cells; *e.b.c.*, external hair cells; *e.s.f.*, external spiral fibres; *i.s.f.*, internal spiral fibres. Note that a large number of the external hair cells are supplied by a single nerve fibre, and that the internal hair cells are supplied one or two at the most by a nerve fibre.

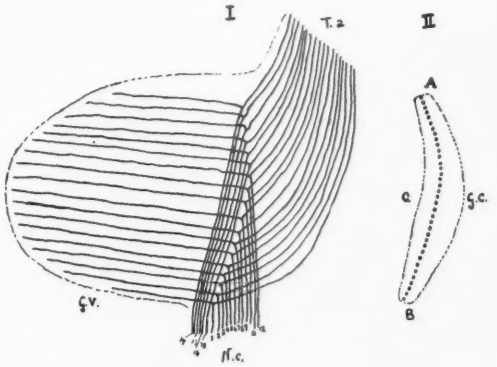

Fig. 4. Diagram reproduced by permission of R. Lorente de No from the article "Anatomy of the Eighth Nerve." Published in the Laryngoscope, Vol. 43, No. 1, 1933. I. Shows the endings of the cochlear nerve in the primary cochlear ganglion of the medulla. II. Showing the arrangement of the nerve endings in the cochlear ganglion and the boomerang appearance with the largest number of nerve fibres at the center.

on the basilar membrane within 10 per cent of our localization. One of our co-workers, Derbyshire,[8] has also obtained evidence of localization in the cochlear nerve. Thus the accumulated evidence at present is in favor of the place perception or the resonance theory, rather than the telephone theory. (Figs. 5 and 6.)

In the course of experiments done to produce deafness by long exposure to a loud tone, evidence was obtained as to a difference in function of the internal and external hair cells.[7 9 17 20] Animals that have been exposed to loud tones for long periods of time showed degeneration of the external hair cells of the organ of Corti and as a result a loss of response from our tests of about 30 decibels (db.). The loss was over a large range but below the tone used in exposure—2,500 cycles per second (d.v.). The damage found was always toward the apex and extended slightly toward the base of the basilar membrane. This loss varied with degree of degeneration, being smallest with the least degeneration and greatest with the most. We also had similar evidence from an animal in which the external hair cells had degenerated from disease while the internal hair cells functioned and appeared normal. Here the response showed a loss of 30 to 40 decibels (db.) throughout the audible range. From the position of the internal hair cells one would expect that its threshold should be high. The internal hair cells rest on bone and therefore are not so sensitive to small movements of the basilar membrane. The external hair cells should be more sensitive to weak stimuli than the internal hair cells, for they rest on the basilar membrane. The external hair cells are probably not concerned so much with the accurate discrimination of pitch as with the detection of weak sounds. The internal hair cells, by their simple nerve supply, should be able to discriminate pitch accurately. This is borne out by the fact that Shower and Biddulph[19] also found that at 5 decibels (db.) above threshold, man discriminates 500 tones against 1,300 at 40 decibels (db.). Of course, one cannot sharply demark one functioning cell from the other. They are always working together. In fact, a tone 20 decibels (db.) above threshold stimulates a considerable part of the basilar membrane. Some principle, such as Wilkinson and Gray's maximal stimulation[23] or the contrast effect of Bekesy's,[1 2] must be used to explain the sensitivity of the ear to small differences of pitch. (Figs. 7, 8, 9, 10.)

In the "nerve deaf" (perception deaf) person, as we observe clinically, the patient is deaf to whispered or conversational voice, but hears and discriminates pitch accurately. The patient attends concerts and enjoys music, though deafened for normal conversation. From the evidence we have at present, we believe that a great ma-

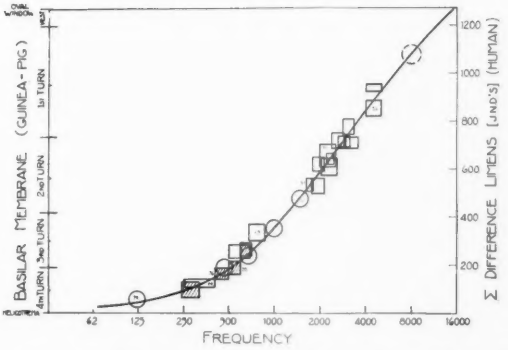

Fig. 5. Curve showing the correlations of the lesions made in the guinea pig's cochlea, the tone loss found, and the curve from Shower and Biddulph's work. Note how closely these two are superimposed on each other. The squares and circles in the figure represent the lesion and the tone loss. The curve drawn represents the integration of Shower and Biddulph's curve for "Differential Pitch Sensitivity of the Ear." Published in J. Acous. Soc. Amer., 3:275-287, 1931.

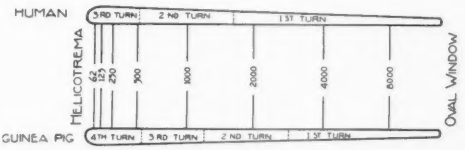

Fig. 6. Shows relative positions of the tones on the basilar membrane in the human and guinea pig. Note the considerable crowding of the tones from 250 to apex, and the crowding of tones towards the round window from 8,000 up.

jority of our "nerve deaf" people are suffering from an end organ disturbance. There does not have to be a complete degeneration of all external hair cells to cause deafness. A diffuse or scattering degeneration of the hair cells throughout the cochlea can be present. This cannot be detected in human pathologic specimens with our present technic. A degeneration of this type might cause a loss of hearing of 20 decibels (db.). Neuritis may cause deafness, but the question always arises as to why does not the discrimination of pitch go as well. It is improbable that neuritis should be able to pick out specific nerve fibers without affecting others. It is asking a little too much of the nerve to degenerate so that there should be a loss of hearing for 20 to 30 decibels (db.) and a fairly normal acting cochlea above this intensity.

To a minimum stimulus the normal nerve responds at its maximum or not at all. This is known as the all or none law. Following an impulse the nerve is refractory for 1/1000 of a second. Masking of sounds depends on this principle.[5 6 8] We have found that masking occurs at the nerve endings in the organ of Corti. The nerve responds to one sound and not to the other, though both are present in the electrical response of the cochlea.

The experimental and anatomic evidence up to the present time points more to the resonance theory of hearing as the correct one. There is spread of sounds on the basilar membrane, but we believe this spread has to do with perception of intensity of sound rather than with that of pitch.

The improvement of hearing in deafness of the so-called nerve type after treatment with various endocrine extracts or bettering of general health can be explained in two ways, apart from purely psychic improvement.

1. An improvement of the reception of nerve impulses in the central nervous system.

2. A restoration toward normal of the organ of Corti, which is necessary for stimulation of the nerve endings.

The loss of hearing of the perception type can be explained as being caused by the following factors:

(a) Degeneration of the receptive organ (organ of Corti).

(b) Lesions of the auditory nerve or central nervous system interfering with transmission of nerve impulses.

Fig. 7. Organ of Corti of guinea pig, 300 x. Exposed to a tone of 2500 cycles per second (d.v.). External hair cells degenerated. Internal hair cell normal though nucleus is out of focus in microphotograph. *EHC*, external hair cell; *IHC*, internal hair cell.

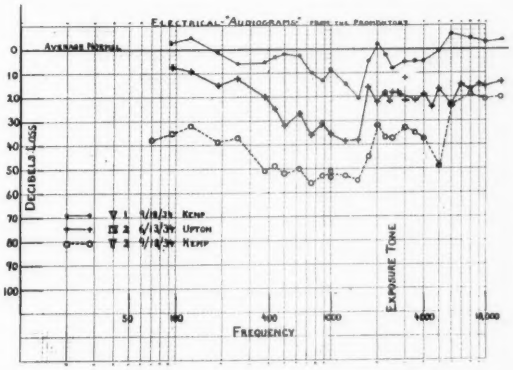

Fig. 8. Electrical audiograms of guinea pigs exposed to a tone of 2500 cycles per second (d.v.). Note the marked loss of low tones below the exposure tone and the slight loss of high tones above this exposure tone. The one showing the greatest loss also had a hemorrhage in the cochlea at the time.

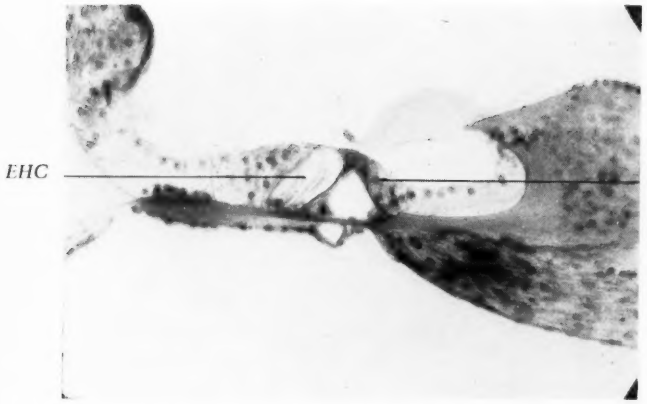

Fig. 9. Organ of Corti of cat, 300 x. Animal with loss of hearing throughout range due to disease. Note the complete degeneration of the external hair cells. The internal hair cell appears normal. Compare with normal organ of Corti and organ of Corti of guinea pig deaf by exposure. *EHC*, external hair cell; *IHC*, internal hair cell.

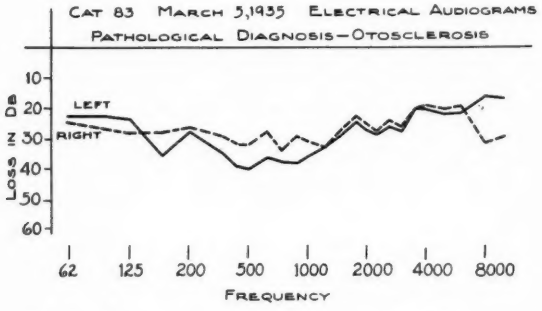

Fig. 10. Electrical audiogram of cat deaf as result of disease showing a loss of 30 to 40 decibels (db.) throughout the range.

CONCLUSIONS.

1. That neuro-anatomic and physiologic evidence is in favor of the resonance theory.

2. Pitch perception is definitely placed on the basilar membrane of the cochlea.

3. There is apparently a difference in sensitivity between internal and external hair cells. The external are responsible for detection of very faint sounds. The internal hair cells are concerned with the fine discrimination of pitch.

4. The external hair cells are more liable to degeneration than the internal hair cells.

5. The chief cause of perception deafness is probably a degeneration of the external hair cells.

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XIV.

THE MECHANICS OF THE MIDDLE EAR PROSTHESIS.*

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It is well known that where the drum membrane and outer ossicles have been removed or lost through disease the patient exhibits a marked reduction in the acuity for hearing air sounds. This deafened condition is at the present time being treated in one of four ways. The first method consists in substituting a compensatory increase in the intensity of the sounds to be heard. This is accomplished by talking loudly to the deafened person; or by amplification of the air sounds through some hearing aid; or by preventing the dissipation of sounds from the source to the ear as in a speaking tube; or by increasing the reflection from the auricle by placing the cupped hand back of the ear or by the use of some device which is based on this principle. The second method takes advantage of the fact that a conduction deafened person may have a normal sensitivity for bone conducted sounds and employs a bone receiver patterned after the original model described by F. W. Kranz and myself[1]; or makes use of the direct conversion of air vibrations into bone vibrations by means of the acoustic fan.[2] A third method seeks to substitute another sense organ for the defective hearing as in lip-reading or in the less effective tactile sensations. While all of these methods may be useful, none of them may be regarded as a direct correction of the physical handicap under which the patient is hearing.

So far as the writer is aware, only the fourth method may be regarded as a direct correction, in that effort is made to replace the lost sound conduction apparatus. This method has been in vogue for many years, possibly centuries. It has undoubtedly been re-discovered innumerable times when a patient in whom the drum membrane and outer ossicles are wanting, has attempted to clean out the external auditory canal and quite accidentally touched some particular area of the medial tympanic wall and experienced an enhancement in acuity of hearing.

In its most familiar form, the middle ear prosthesis is an oiled or moistened cotton pellet. This pellet, when applied to the proper

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area on the medial tympanic wall, sometimes referred to as "the hot spot," may produce a subjectively marked increase in the acuity for air sounds. Many of these cases may show referred symptoms such as nystagmus, vertigo and the like, while others experience a sensation of "things opening up" or a feeling of fullness in the face. The position of the area affected by the prosthesis and the reason for the increased acuity in hearing have been subject to no little speculation and also to some direct experimentation.

It is very difficult in the usual case to make direct observations on the position of the area affected. The external auditory canal is too small and the cotton pellet necessarily intervenes between the contact point and the eye of the observer. It is for this reason that the patient is usually more adept in placing the prosthesis in position than is the otologist. The usual reactions, however, indicate that the prosthesis exerts pressure upon some relatively movable area directly related to the labyrinth liquid. This limits the contact area to the oval or the round window. The oval window, containing the footplate of the stapes, is in a nearly direct line with the axis of the external auditory canal. This contact area is therefore more accessible than is the round window, which is normally hidden behind the promontory and at the bottom of a fairly well defined niche. However, whether the prosthesis is applied to the oval or to the round window, the effect should be quite similar. Many otologists appear to be convinced that it is the round window which must be contacted, but this is contrary to the evidence, as will be pointed out later.

Two papers dealing with this problem were presented at the meeting of the Association of American Anatomists in 1932. These two papers indicated two entirely different interpretations on the physical behavior of the prosthesis in its relation to the internal ear. The one interpretation was presented by Hughson,[3] on the basis of extensive researches by Hughson and Crowe, at the Johns Hopkins University Otological Research Laboratory. The other interpretation was offered by the writer,[4] as a result of limited experiences in the quantitative reactions found in a few cases of ossiculectomy.

Hughson and Crowe employed the Wever-Bray technic in cats and found there was an appreciable or even a marked enhancement to the sensitivity of response when a moist cotton pellet was applied to the round window membrane. They interpreted this result to mean that the prosthesis stabilized the round window membrane and thereby reduced its efficiency as a sound absorbing structure. The writer's interpretation was that the cotton pellet vibrated and acted as an inefficient transformer in the conduction of the air vibrations

to the cochlea. It is at once apparent, whether the effect may be due to a stabilization of the round window (Hughson and Crowe) or due to vibratory reactions (Pohlman), there must surely be some device available which is more effective than an oiled or moistened cotton pellet. Logically if the cotton pellet produces an increase in the acuity for air sounds, then if it is to be replaced by something more efficient one must know how this increased acuity is brought about. This is true because a stabilization of the round window membrane is an entirely different mechanical problem than that of increasing the vibratory responses in this structure.

The preliminary researches by Hughson and Crowe[5] led them to consider the possibility of replacing the cotton pellet by something which would be at least more permanent and at the same time be more acceptable surgically than a loose cotton mass which undoubtedly sheds in the middle ear cavity. They found by experiment that it was possible in cats to place a connective tissue implant on the membrane of the round window.[6] This graft took well and seemingly was just as effective, if not more so, than the cotton pellet. While Hughson and Crowe avoid all mention of the applicability of this procedure to a deafened human being, there can be little doubt that this possibility might occur to anyone reading the record of their extremciy careful investigation. However, before so serious an operation may be performed on the human being, there are a number of points which must be satisfactorily established. The first point implies a more definite knowledge on how the prosthesis operates and whether or not a connective tissue implant on the round window membrane is the most efficient treatment. The second point, which must be considered in carrying these experiments on cats over to the human being, is to establish that the enhancement of the Wever-Bray effect in cats is an absolute criterion of the acuity of hearing which might be conferred on the human being. The third point, which can only be decided later, is to determine that the amount of enhancement to air acuity conferred is worth the risk involved in the operation. After all, the patient is not interested in the theoretical aspects of the problem. What he wishes to know is how much increased acuity he will enjoy if the operation is successful and at least something about the risks entailed.

It is evident to anyone oriented in the anatomy of the region that placing a fascial graft upon the membrane of the round window of a cat is a relatively simple affair when compared with a similar operation in the human being. Not only is the human round window area relatively more inaccessible, but the operative manipulation nec-

essary to place a graft, once the region is exposed, is in itself a highly refined bit of surgical technic. The operative difficulties are so great that it is essential to establish all information on a definite scientific basis. This is especially true when there seems to be some difference of opinion on the behavior of a prosthesis. Accordingly, it is only fair to the problem, if one is interested in its theoretical aspects, and only fair to a prospective patient, if a practical application of the Hughson-Crowe experiment is contemplated, that these differences in opinion be discussed frankly.

Hughson and Crowe sum up the evidence in the following words:

> "In the preliminary report of this investigation the following statement was made: "The secondary tympanic membrane apparently acts as safety valve to protect the structures of the inner ear and owing to its mobility absorbs a large percentage of the sound impuses that reach the cochlea." There appears no reason for changing this view at the present time. Obviously pressure upon the membrane cannot materially affect intralabyrinthine pressure more than momentarily. Indeed, there is already experimental evidence at hand indicating that greatly increased intralabyrinthine pressure has no effect whatever upon the transmission of sound. There remains then, no other explanation of the phenomenon but that of immobilization or fixation of the round window membrane itself."

This statement of the interpretation is concise. It is unfortunate that Hughson and Crowe did not elaborate on it in greater detail, because the meaning is by no means as clear as they may have supposed. The "safety valve" comparison may not have been particularly well chosen, because a safety valve, in the usual acceptance of the term, implies a pressure regulating mechanism which is tripped at a given pressure. Similarly, to state that "the round window membrane by reason of its mobility, absorbs a large percentage of the sound impulses that reach the cochlea" requires a further statement to explain how this may come about. Sound absorption usually means that the energy in the vibrations is transformed into terms of something else like heat. Accordingly, the prosthesis on the round window membrane might, through its fixation, prevent the air sounds from reaching the cochlea, but there is no evidence that air sounds may enter the cochlea effectively by this route. We are then forced to the conclusion that the mobility of the membrane absorbs the sound impulses after they reach the labyrinth liquid. How can the membrane act as a proper shock absorber when it is placed on the wrong side of the end organ? In other words, after the sound impulses have affected the end organ, how may they be absorbed by the round window membrane? If the round window membrane acts in opposite phase to the stapes foot plate, and if the motional reaction in this membrane is artificially limited, then the amplitude of response in the labyrinth

liquid will be correspondingly decreased and the pressure increased. Does the evidence therefore show that as the motions in the liquid are decreased the acuity for hearing air sounds is increased? Clearly then the way out is to abandon the accepted idea of displacements in the liquid of the internal ear as the mechanical means of activation and substitute for this the doctrine that the end organ is activated by pressure. However, this statement would imply that the round window is the only area which reacts to pressure, which is contrary to the evidence submitted by Bezold[7] as a result of his experiments with a labyrinth manometer. There are others, notably the writer, who believe that any and all of the possible pressure compensation areas are too slow in their response to have any relation to the physics of the end organ activation. Accordingly, the interpretation as offered by Hughson and Crowe may still be considered within the realm of controversy. The statement "that there remains then, no explanation of the phenomenon but that of immobilization or fixation of the round window membrane iself" is emphatic but it is by no means convincing.

In the preliminary paper published by these investigators, it was reported that filling the niche of the round window with plaster had no appreciable effect, while the application of the cotton prosthesis to the round window membrane resulted in a definite enhancement of the Wever-Bray effect. The original interpretation of this result was that the application of the cotton prosthesis to the round window membrane created an optimum pressure in the labyrinth which in turn accounted for the result. However, this interpretation has been changed to the decrease in the sound absorbing function of the round window membrane due to its immobilization. My own comments at the time of the preliminary report were[8]: "There is no reason to suppose that a cat with the niche of the round window filled with plaster will not have the normal optimum labyrinth pressure, but a plug of this character will not vibrate as readily as a moist cotton pellet; hence the negative result." The evidence at hand, therefore, does not substantiate the claim that the cotton pellet actually immobilizies the round window membrane but indicates where the prosthesis vibrated, the result was positive; and where it did not, the result was negative. So far as the results obtained through the Wever-Bray technic are concerned, they might have been quite similar, according to either explanation, and thus far nothing may be said to have been proved definitely one way or the other.

This brings up the second question as to whether the Wever-Bray effect is to be regarded as an absolute criterion of the sensitivity

of the end organ as expressed in terms of acuity of hearing. The writer has had no personal experience with this technic and therefore quotes from an investigator who has considered this point in relation to the facts as determined in the human being. Stevens,[9] in his paper on "The Relation of Pitch to Intensity," says in conclusion:

> "The failure of pitch to exhibit a one-to-one correlation with frequency means, on the basis of a resonance theory of hearing, that the point of maximal stimulation on the basilar membrane due to a given frequency, changes position with changes of intensity. It seems certain that one is correct in attributing the change in pitch to a peripheral mechanism and in particular to the spatial pattern of excitation in the cochlea, since an examination of the electrical impulses recorded from the auditory nerve of cats and guinea pigs has repeatedlly failed to show a change in the frequency of the impulses when the intensity of the stimulation is increased. For all except the high frequencies nerve impulses continue at all intensities to have the same frequency as that at which the ear is being stimulated. Therefore the possibility that the pitch of the tone is determined by the frequency of the impulses reaching the brain is definitely excluded, and with it the so-called "telephone" or "frequency" theories of hearing. It would seem in fact that the phenomenon of synchronized impulses in the auditory nerve (the Wever-Bray effect) is an interesting effect useful in throwing light upon the nature of the activity of the cochlea but having nothing to do with the determination of that discriminatory response which we call pitch. It is probably a case of a correlation without a causal connection."

There is accordingly a reasonable doubt that the Wever-Bray effect is synonymous with acuity of hearing. It must, however, be admitted that if there is a correlation but not necessarily a causal connection, the technic might still prove useful in the determination of the absolute hearing acuity in experimental animals. In any event, the only check is in terms of the human being where the Wever-Bray effect cannot very well be elicited but where the absolute acuity in hearing may be readily determined. The question may therefore be stated as follows: "Is it possible in the human being to determine whether the prosthesis applied to the medial tympanic wall stabilizes the window to which it is related or vibrates the area of the window contacted?"

If it can be proved that stabilization of the window is the cause of the increased acuity for air sounds, then the Hughson-Crowe interpretation is correct. If, however, it can be proved that the increase in air acuity is dependent on the vibrational reactions in the window contacted, then the Hughson-Crowe interpretation is incorrect. In either case, the Wever-Bray technic may still be useful in the determination of responses in experimental animals.

The explanation proposed by the writer was that the prosthesis vibrates and acts as an inefficient transformer to conduct the air

vibrations into the labyrinth liquid. It must be admitted that a cotton pellet, oiled or moistened, does not look as if it could have much of a vibratory reaction. In point of fact Hughson, who knew of this interpretation, dismissed it as a possibility. The writer has repeatedly called attention to the futility of attempting to see how one hears and has compared this type of visualization to listening at a dead eye with a stethoscope to hear how it may have seen colors. The interpretation of a vibratory response was not merely guessed at, but came out of the experimental evidence that a light prosthesis produces a different sensitivity curve than a heavy one.

We are, therefore, driven back to the human being as a court of last resort and to the type of human being who may profit in the application of such directed research. Fortunately, the writer located in Omaha another Alexis St. Martin in the person of a patient whose condition and intelligent co-operation made this preliminary report possible. This patient was operated upon some fifteen years ago by Dr. H. I. Lillie, and a complete radical mastoidectomy was performed on the right ear. Some years later a similar operation was indicated on the left and was performed by Dr. Claude Uren of Omaha, through whose courtesy the patient was referred to me. Both middle ear cavities are epidermatized and dry. The acuity for hearing air sounds is about the same on the two sides. The right ear seemingly required a more extensive operation than the left because the right canal is considerably widened. It is possible to inspect the medial tympanic wall on this side without introducing a speculum. The medial opening of the canal is somewhat crescentic, with the concavity downward. The round window area is completely hidden. The bulge of the promontory is easily identified and superior to it the wall shows a slight depression about 2 mm. wide which occupies what would be the normal position of the stapes. The depression is probably in part due to the overhang of the processus cochleariformis. The lining membrane is somewhat thickened as is usual in such cases.

The patient employs an oiled pellet as a prosthesis. This he places into position, judging the efficiency of the placement by the sound made by snapping the fingers. He is definitely Rinne negative, both with and without prosthesis, and in no instance has an average enhancement to air acuity through the speech range measured more than 10 db. The increased sensitivity is more marked in the upper frequency range, and this does not appear to be uncommon, because it was found in the case reported by the writer[10] and also in the one tested by Lillie and Kranz.[11]

The first experiments consisted in substituting a prosthesis of lamb's wool covered with animal membrane for the cotton pellet. While this lighter and firmer substitute showed the same effects as those noted in a previous case, the added sensitivity was considered of negligible importance. He is deafened without prosthesis, as determined by the Western Electric Co. 4a audiometer by a factor of 54 db. and with cotton pellet in place by 48 db. These readings were taken with the receiver provided with a large rubber cup. This cup materially increased the amount of air trapped and required a correction for a normal reading of 9 db., or three steps. The rubber cup, however, quite completely ruled out all possibilities of a direct bone conduction due to the vibration of the receiver as a whole—a factor which must be considered in intensities of this order. Subjectively the increase in air acuity by a factor of 6 db. is of material benefit, but even with the pellet in place the subject is as deaf as the figures and the definitely negative Rinne report would indicate.

It was thought necessary to find out what would happen to the acuity for hearing air sounds if the stapes area was immobilized beyond the fixation caused by being buried in the thickened lining of the middle ear. It must be noted that in cases of this sort the acuity of hearing is somewhat better immediately after the operation and when the reaction in the lining membrane takes place, the sensitivity to air sounds is somewhat reduced. These cases also show lowered acuity for air sounds when the middle ear cavity is quite dry over that found when the lining membrane is moistened or oiled.

A delicate probe of bamboo, about the diameter of the ordinary sewing needle, was tipped with a small bead of dental impression compound. It was found that a relatively small bead was more effective than a larger one and in no case must it exceed the stapes area. The patient held this probe between thumb and first finger and introduced it until it touched the "hot spot." It was unnecessary to caution him to be careful and gentle. On inspection the tip of the probe could be seen resting against the stapes area, and the subjective sensations were that the tip was in place. However, no enhancement to acuity for air sounds was noted, in spite of the fact that now the stapes was more or less stabilized. Blowing gently across the projecting probe was described as a "roar," and when the stem of a vibrating fork was applied to the knuckle the sound was heard as "loud." The results seemed to indicate that the effect was not due to a stabilization of the stapes but to the vibrations which passed through the probe.

A second series of experiments were undertaken following the physics of the diaphragm and rod, as described by the writer in 1922.[12] A preliminary test was made with a diaphragm placed over the wide end of an ear speculum. A tipped bamboo rod, of sufficient length to touch the medial tympanic wall when the speculum was introduced, was affixed to this diaphragm. The narrow end of the speculum was reinforced with a bit of soft paraffin which could be moulded when the device was in place. The patient introduced the speculum and when proper contact was made he immediately showed signs of a pronounced increase in air acuity because now he was aware that people were talking in the adjoining room. He remarked "I didn't know how deaf I really am." The Rinne test was now definitely positive. Touching the diaphragm gently eliminated all the effect.

This experiment indicated we were on the right track and various type of prosthesis were tried. The latest development was constructed with the technical assistance of Dr. Frank Viner, Dean of the Creighton University School of Dentistry, whose courtesy and ingenuity will be more completely acknowledged in the final report. In this prosthesis, a female element is carefully fitted into the external auditory canal, and the male element, consisting of a diaphragm and rod, may be adjusted into position by the subject. When this prosthesis was in place the patient declared it was noisy. It was found that this noise was due to a lighted Bunsen burner, some eight feet away. Turning off the burner eliminated the noise. Without prosthesis the patient must come within a foot of the burner to hear it. The ordinary conversation was now declared to be loud, which is a relatively normal reaction, because one does raise the voice when dealing with deafened people. A check was made on the 4a audiometer under the same conditions as in previous test, and the hearing for speech was now 15 db. Roughly this enhancement to air acuity by some 40 db. means a change in intensity requirement to about one-ten thousandth of the amount without prosthesis. Inasmuch as no one with a hearing disability of only 15 db. consults the otologist because of an appreciable hearing defect, it is safe to assume that our patient with the device in place would never be suspected of being even hard of hearing, let alone distinctly deaf. Clapping the hands at some five feet distance was noted as very loud but not painful. Placing the eraser tip of a pencil gently against the diaphragm eliminated all enhancement effect.

While this device is patterned after the mechanics of the bird's ear, it still lacks the essential shock absorber in the structure of the columella, as demonstrated in 1914.[13] Inasmuch as this diaphragm-

rod displayed an efficiency which is about that of a normal drum membrane and ossicle chain (if one disregards the added fixation of the stapes by being buried in the thickened lining), it may be necessary to add this shock absorbing quality, which will necessarily reduce the efficiency of conduction somewhat. The details will appear later.

It is apparent that in these experiments where the stapes area was definitely stabilized, no enhancement in acuity for air sounds was experienced. It is also obvious that when the stapes area was vibrated a pronounced improvement in acuity for air sounds obtained and where these vibrations were damped through eliminating the vibrations in the diaphragm all enhancement was lost. In point of this fact, in the first series of experiment the rod was introduced after the diaphragm was in place and then sealed to the diaphragm with wax. The patient could hear a progressive improvement in acuity as the wax cooled and integrated diaphragm and rod. This experimental evidence quite definitely opposes the interpretation by Hughson and Crowe, but by no means eliminates the usefulness of the Wever-Bray technic in its relation to experimental work in animals. Probably a definitely vibrating prosthesis, such as is briefly described here, will show the same effect when placed in contact with the round window area in cats. In any event, the increased acuity to air sounds when the vibrating prosthesis is brought into contact with the stapes footplate might also be experienced if the round window region was accessible and the membrane sufficiently thickened to justify the application of the device.

There is no reason to suppose that the extremely light prosthesis under construction may not be worn with comfort or that there will be untoward reactions resulting from the delicate contact between the gold footplate and the stapes area. It is also suggested that in cases of complete radical mastoidectomy the field of operation be left so that a high efficiency prosthesis may be easily applied. The inconspicuousness of this device, unlike that of the usual aids to hearing, will not contribute to its relative inefficiency, and the diaphragm should be located below the level of the tragus to insure reflections of the sound pulses at the auricle. Naturally it would be just as impossible to produce a standard device which would fit all ears any more than it would be possible to develop a dental prosthesis which would fit any mouth. However, the mechanical unit in its final form should be easily adapted to any particular case.

We feel that sufficient evidence has been submitted to show cause why we differ with the statement made by Hughson and

Crowe, namely: "There remains then no other explanation of the phenomenon but that of immobilization or fixation of the round window itself." There still remains the other possibility suggested by the writer in 1932, namely, that the prosthesis vibrates, and the usual prosthesis of cotton acts as an inefficient transformer in conducting air vibrations to the labyrinth liquid. There is no direct evidence that a fascial transplant on the round window membrane behaves physically in the manner suggested by Hughson and Crowe or that it will necessarily be any more efficient than a cotton pellet. Indeed Oppikofer[14] reported two cases in which the round window niche was filled with adipose tissue and no appreciable effect on the acuity of hearing was noted.

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XV.

SOME SPHENOPALATINE SYNDROMES: A REVIEW OF SLUDER'S OBSERVATIONS ON THE SPHENOPALATINE GANGLION.*

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It seemed that a summary of Dr. Sluder's clinical study of the sphenopalatine ganglion would afford an interesting background to the papers to be presented today, as well as a slight tribute to one who had contributed so much to our specialty as a clinician and a teacher.

Among his numerous publications I have found thirty-five devoted to this particular topic. The large proportion of papers on this subject, by one who never wrote unless he had something definite to say, and which he thought would add to our knowledge of otolaryngology, gives us some idea of how important he considered this problem, and of the pleasure its continued study afforded him.

A perusal of his writings impresses one with certain characteristics which are typical of the man as we knew him: First, his painstaking, accurate and long-continued observation of the clinical symptoms of his patients and their behavior under various methods of treatment, and his careful checking of these before publication. His own accuracy of observation may, at times, have led him to accept as authentic the reports of others who were less exacting in their methods. Second, the constant study of his numerous specimens in an effort to establish a solid anatomic foundation for his clinical findings, eight of these papers being essentially anatomic contributions. And, third, his many conferences with otolaryngologists, ophthalmologists, neurologists, physiologists and pathologists, in an effort to evolve a rational explanation for the various phenomena he observed.

In his first paper[1] on this subject, in 1908, he said: "Occasionally it has been my lot to be consulted by patients who were in every way healthy and normal, but who suffered much of the time from headache which did not follow any of the known rules. It was irregular

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as to the time of its appearance and irregular as to the part of the head involved. Sometimes it was referred to behind the eyes, sometimes to the upper jaw in front, and sometimes to the hard or soft palate. Occasionally the nose was said to ache in the back, or the teeth were described as sore, or this pain was referred to the temple and to the occiput or neck. It sometimes would become severe and behave as a migraine. Sometimes an indefinite sense of stiffness was described. These symptoms appeared from day to day, alternating one with the other, or associated two or three together. On one occasion I had them all described at once.

"Within the past six months I have seen some high grade inflammatory troubles in the posterior ethmoid and sphenoid sinuses, which have subsided and healed, usually within a week or two, but have left the train of symptoms, as described, in their wake, and these have continued on more or less indefinitely. This exceedingly irregular train of symptoms cannot be explained on a basis of inflammatory troubles in the accessory sinuses.

"They seemed to me neuralgic and to have been excited by the forerunning inflammatory process in the nose.

"The position of the sphenopalatine ganglion in such close relation to the accessory sinuses and the external wall of the nose suggested the possibility of its involvement, secondary to inflammations in those parts. Furthermore, the distribution of the nerve processes of this ganglion corresponds closely with many of the regions to which pain is referred by these patients.

"I then began the experiment of anesthetizing the ganglion (with cocain). The relief would last for from fifteen minutes to an hour. Occasionally it would abort the attack.

"Of the ten cases I have observed, none were of sufficient severity to warrant operative interference. Application of silver, 2 per cent, or formaldehyde, one-half per cent, to that area either helped or cured the pain."

In 1909,[2] he stated that none of the descriptions of the sphenopalatine ganglion in the sphenomaxillary fossa in the anatomic textbooks suggested any close relation of this structure to the nose or its accessory sinuses, and that the special treatises on the nose made no mention of such relation. He presented anatomic specimens showing this close relationship, the ganglion sometimes lying within one or two millimeetrs of the nasal mucous membrane, although at times it might lie as deep as seven to nine millimeetrs. However, it always lies close to the top of the fossa. He also called attention to the fact

that the current descriptions of the fossa gave the impression that it was surrounded by solid bone, and made no mention of the fact that the bony walls separating the fossa from the nasal sinuses were very thin, and said:

"It seems essential that in any comprehensive description of these parts, special emphasis should be laid upon their intimate relations to the nose and its accessory sinuses; and this is certainly true if the anatomy is to lead the way to clinical application or investigation."

In making a clinical application of these anatomic facts, he says:

"It has long been recognized that inflammatory processes in the sphenoidal, postethmoidal and maxillary sinuses extend to the optic nerve, and this fact has been demonstrated postmortem. It is therefore altogether reasonable to assume that these processes also pass over to Meckel's ganglion, although the picture is very much less striking than the blindness produced by involvement of the optic nerve.

"According to my observations, characteristic disturbances have followed postethmoidal and sphenoidal inflammations, which cannot be explained otherwise than by assuming that the sphenopalatine ganglion has become involved by extension. Some of these disturbances have been transitory and some have persisted for many years."

After an observation of forty-seven cases, he now gave the following description of the syndrome as the picture oftenest presented: "Pain which begins at the root of the nose, extends downward over the maxilla and backward in the mastoid to become severest about five centimeters posteriorly to its tip; thence extending backward to take in the entire occiput, and downward into the neck, shoulder blade, shoulder, and sometimes into the axilla. With the severest attacks it extends down into the arm, forearm and even to the fingers. According to my observations, this pain very rarely invades the upper part of the head. When very severe it may extend a little way into the brow or somewhat above the line of the zygoma. I have seen this picture complicated by or associated with other headaches; but the distinctive 'neuralgic phenomena,' as outlined before, and which have proved uniformly amenable to treatment (cocainization of the nose) have not extended to the upper part of the head." (From this he derived his term "lower half headaches.")

He called especial attention to a case of acute glaucoma, reported by Dr. A. E. Ewing.[3] "One application of one drop of 50 per cent

cocain to the sphenopalatine foramen stopped the violent pain. It did not return." He emphasized the fact that the idea of associating glaucoma with Meckel's ganglion was Dr. Ewing's.

In a communication before the Section for Internal Medicine of the St. Louis Medical Society, January 15, 1910,[4] he said:

"I have since seen two cases of typical sphenopalatine neuralgia in which there was no evidence of previous sinus inflammation. The pain was apparently a typical migraine, and was stopped by applications of cocain to the nasal mucous membrane at the site of the sphenopalatine foramen, beneath which the ganglion lies."

In a paper[5] read before the American Laryngological Association, May 3, 1910, after again calling attention to the close relationship of the ganglion to the postnasal sinuses and to the fact that in common with all sympathetic ganglia it has a motor, a sensory and a sympathetic root, and that "some gustatory fibers seem also to pass through one of the roots," he says: "It is not surprising that the clinical manifestations arising from disturbances of this nerve center should be multiform."

Based on a complete investigation of sixty cases (his earlier observations having been directed merely to the painful manifestations), he formulated a syndrome of these disturbances:

The *Neuralgic Syndrome* corresponds very closely to his description in 1909.[2] He also mentions that these neuralgic manifestations may begin to run irregularly, assuming the form of migraine, which may persist for years after all local inflammatory conditions have disappeared.

He also describes:

Sensory Signs.—An accompanying slight anesthesia of the soft palate on the affected side, and of the pharynx as far down as the lower part of the tonsil; also a slight anesthesia in the lower anterior part of the nose.

Motor Signs.—The palatine arch is often, but not always, higher than on the well side. The uvula is often deflected from the affected side.

In the movement of the soft palate in closing off the nose from the throat, the median raphe is deflected from the affected side, and the dimple which forms just above the uvula during this act is displaced to the well side. Normally it is in the median line.

Gustatory Changes.—Tests made (with minute droplets of solutions of sugar, citric acid, sodium chloride, and bisulphide of quinine applied simultaneously on both sides of the dorsum of the tongue in its middle third) showed in almost every case a diminished and delayed perception of taste on the affected side. Three cases, just before or during an attack of migraine, showed a perverted sense of taste.

He reported the results of his experiments to determine whether the application of cocain to the region of the ganglion was essential, or whether applications of cocain to any point in the nose might not also give the same result; also whether the relief might not be due, in part at least, to the exhilarant toxic effect of the cocain.

He sums up as follows: "When other areas were cocainized the result was negative. When the sphenopalatine area was cocainized the pain stopped."

In a paper[6] published in 1909, in the Journal of the American Medical Association, he states that the injection of alcohol into the sphenopalatine ganglion, which he had adapted from this treatment of peripheral nerves by the injection of the nerve trunks, did not give the satisfactory results reported by others in other types of neuralgia. The injection was exceedingly painful, and this often lasted for several days, even though the ganglion had been previously cocainized, and the relief was given only for a short period of time, sometimes for as little as two weeks.

After experimenting with combinations of alcohol with chloroform, novocain, cocain and eucain to alleviate the pain of the injection, he found that phenol, added to the alcohol, diminished the pain and that when the strength of the phenol was raised to 5 per cent the injection of one drop into the ganglion was painless and apparently curative for all grades of this neuralgia.

He reported the use of this fluid in seven cases of sphenopalatine neuralgia and three cases of glaucoma, with no return of pain since the injection—the longest elapsed time being ten months.

In 1912[7] he calls attention to the fact that if, when alcohol is injected into the ganglion, the needle is placed too far internal, the pain produced is projected backward (along the vidian nerve), and when placed too far external, the pain is referred to the upper jaw (second division of the fifth nerve); but if the injection is directly into the ganglion the pain is referred in both directions.

He also states that inflammation in the sphenoid may present a picture identical with neuralgia which starts in the ganglion, and offers the anatomic explanation that the nerve trunks going to the ganglion (the vidian and the branch from the second division of the fifth nerve) may, by reason of their proximity to the sphenoid, become readily inflamed in sphenoiditis. The differential diagnosis can be made by the fact that cocainization of the ganglion controls the pain when it is originated in the ganglion, but fails to stop it if it originated in the sphenoid, because the ganglion is peripheral to that point of origin. However, cocain applied within the sphenoid does not stop the pain in the latter type of case.

He cites an illustrative case of vasomotor rhinitis with paroxysmal sneezing, profuse watery discharge, and asthma, in which all intranasal measures were unavailing but which yielded to intrasphenoidal applications, offering the above anatomic explanation.

In 1913[8] he restated his belief that many (but not all) of the recurrent headaches that bear the name "migraine" are sphenoidal empyemata, or were started as such, and that the nerve trunks have become involved by extension of the inflammation (or its toxin) through the thin layer of bone intervening, and the cases he has seen strengthen his belief in the correctness of his previous deductions He feit, however, that at least five years should elapse before definite conclusions should be drawn.

In 1913[9] he reported a series of twelve cases of systemic toxic origin, patients well known to him, where there was no preceding localized inflammation or coryza. Cocainization of the ganglion in these cases always controlled the pain, and he describes in detail his technic for injecting the ganglion. By this time he had increased the amount of phenol alcohol to .5 cc.

In a paper[10] read before the Mississippi Valley Medical Association, Cincinnati, Ohio, October 27, 1914, he uses the term "nasal ganglion" as synonymous with "sphenopolatine" because of its greater appeal to the clinician. He further states that the pain of corneal ulcer, iritis and interstitial keratitis can be controlled by cocainization of the ganglion.

In recording accidents, he says that in nearly five hundred injections he encountered hemorrhage twice, secondary bleeding (five days after) once, and paralysis of the abducens once (the squint lasted eight weeks; the diplopia cleared up in twelve weeks).

He injected one case in which cocainization of the ganglion increased the pain. The injection made the condition worse.

He then describes at length the sympathetic syndrome, stating that: "The neuralgic syndrome is sometimes variegated by the addition of symptoms of a vasomotor and secretory type, and sometimes it happens that these are the only symptoms of disturbance in the nasal ganglion.

"They may be aroused by a slight coryza that has had little time for development, and appear with explosive effect, out of proportion to the pathologic lesion. The patient in health is for the first time, regardless of the season of the year, suddenly seized with severe and protracted sneezing, accompanied by such nasal congestion and thin, hot secretion, so profuse as to have him resort to a towel to use as a nose-cloth. With the great congestion of the nasal fossæ is seen great redness (congestion) and swelling of the external nose, and soon thereafter appear more or less roughening of the skin from the secretion and the wiping it necessitates. In addition to these symptoms, the eyes are greatly reddened (congestion of the conjunctiva) and bathed in tears of a profusion corresponding to the nasal secretion, accompanied by dilatation of the pupil. They are involved almost, if not quite, simultaneously with the nose. A sense of itching or burning or a feeling of wind blowing into them accompanies the lacrimation, together with a feeling of discomfort of a peculiar kind, which seems independent of the secretion and congestion, and there may be photophobia. Work requiring near vision is usually very difficult or not possible under these conditions. Sometimes these symptoms are accompanied by dyspnea with dry räles (asthma).

"For a number of years I have thought that these were symptoms on the part of the sympathetic which supplies the nasal ganglion, and felt that cocain could be used to control them. Various other thoughts prevented me from drawing conclusions from cases of longer standing (vasomotor rhinitis, hay fever, paroxysmal sneezing, rhinitis nervosa). During the past year, however, I have had these cases so acute and so severe that they seemed perfect for the experiment. The nasal ganglion was cocainized. The effect was to quickly stop the secretion in the nose and eyes and bring them back to normal appearance. The swollen, red nose (rosacea) and the puffed, red, disabled, tearing eyes, became normal, and likewise the nasal fossæ. Further experience revealed that these cases in their behavior to cocain were not unlike the painful ones arising from the ganglion. When the case lasts a little longer, the paroxysms begin to appear irregularly and often settle down into the morning sneezing of "rhinitis nervosa." The clinical course of a given case may be of the sympathetic type pure, but upon questioning closely it is very often found that a low-

grade ganglion neuralgia complement exists. Following these cases further, it appears that this syndrome is subject to subdivision, just as the neuralgic syndrome becomes subdivided and betrays itself as single symptoms. So it happens that the sympathetic symptom-complex may present the single symptom—sneezing—or thin secretion, or the eye disturbance which simulates a low-grade conjunctivitis with asthenopia.

"It is generally admitted that the functions of the sympathetic nervous system, among others, are vasomotor and secretory. Sensory attributes have also been proven for it.

"The anatomic and physiologic problems of the autonomic nerves (sympathetic) are very complex, and a clinician may well hesitate approaching them, but it would seem that the knowledge of the present on this subject permits the thought that it is a lesion of the sympathetic elements of the nasal ganglion that explains not only these vasomotor secretory phenomena but also the pain referred into the neck, shoulders, arms, etc.,—the vidian neuralgia. Also, the thought arises that it is the anatomic connection of accelerator fibers for the heart and vasomotor fibers for the lungs, with the ganglia through which the cervical sympathetic passes (lower cervical and first thoracic) that explains some of the cases of asthma of nasal origin. Much more physiologic fact is needed before such conclusions can be asserted. I have been unable to find any statement of what influence is exerted by ganglion cells over nerve fibers in their passage through a ganglion with whose cells they have no other relation. Some influence must be assumed to exist for the above hypothesis. It also would appear to a clinician to exist."

In 1912 and again in 1913 he had stated that he believed the underlying pathology in these cases was hyperplastic sphenoiditis or ethmoiditis, although some of these cases were not such, according to their clinical behavior, and bone removed from some of these patients was declared to be normal by Dr. E. L. Opie.

During the succeeding ten years he presented numerous papers based on individual symptoms, and consolidating his theories.[11-28]

In his presidential address,[29] before the American Laryngological Association in 1926, which, unfortunately, he was unable to deliver in person because of the sudden illness which attacked him while he was on his way to Montreal, he said:

"The anatomy and physiology of the involuntary nervous system are so complicated, and the literature so extensive, that they are

impossible of full comprehension by a rhinologist, busy in practice. Clinical observation, however, is a field open to the humblest of us and must needs be a matter of routine; and each of us must speculate at times in an effort to understand or explain some of our observations. The pathology of the involuntary system is so little known as to be negligible. This system is superficial in the nose and nowhere else in the body. Here it is available for experiment and treatment. This fact may excuse a rhinologist's approach to some clinical features concerning it."

He then mentioned a symposium on Jonnesco's procedure of the removal of the cervical ganglia for the relief of the pain of angina pectoris, at a combined meeting of the Surgical and the Nervous and Mental Diseases Sections of the American Medical Association, where it was brought out by eminent anatomists and physiologists that the procedure could not succeed, but, when it was discussed by surgeons, they reported cases on which they had operated and obtained the desired results. He also called attention to the relief of spastic paralysis by ramisection, as described by Royle, which was discussed by eminent neurologists and surgeons and shown that it gave no relief, yet Royle got the result and so did Adson; also, a symposium on periarterial sympathectomy for Raynaud's disease, where a similar difference of opinion was brought out.

He said: "The obvious fact brought out by these discussions is inconstancy on the part of the sympathetic system and our insufficient knowledge of it. I do not think it can be construed that any one of these accomplished observers is wrong."

He discouraged attempts at the surgical removal of the ganglion, because of the danger of cutting the internal maxillary artery, and because sphenoid lesions could still produce the same phenomena by irritation of the maxillary and vidian nerves, which could not be excised.

He stressed the neuralgic syndrome, or lower half headache, the sympathetic syndrome and the association of the ganglion with ophthalmic migraine, which he considered to be clinical entities.

He admitted that the explanation of the many and sometimes bizarre conditions that are reported as being controlled by cocainization of the ganglion is difficult and sometimes impossible because of the complexity of that structure as well as the part other nerves may play in the clinical phenomena.

His last communication, "Injection of the Nasal Ganglion and Comparison of Methods,"[30] was an anatomic presentation showing

the dangers accompanying injection of the sphenopalatine ganglion through the posterior palatine canal. He presented anatomic specimens with a needle *in situ* demonstrating that if the canal is large or curves slightly outward, the needle may miss the face of the sphenoid and enter the orbit through the infra-orbital fissure and reach the structures in the supra-orbital fissure, the third, fourth, fifth and sixth nerves, and the ophthalmic vein, which is the beginning of the cavernous sinus and close to the optic nerve. He also called attention to the insecurity of relying on measurement of the depth of insertion of the needle, because of the great variation in the length of the canal and in the distance of the floor from the roof of the nose.

His two books, "Headaches and Eye Disorders of Nasal Origin," published in 1919, and "Nasal Neurology," published in 1927, are too well known to require extended comment. They illustrate in a striking manner, however, his intense interest in the neurologic phase of rhinology and the part played by the sympathetic system.

The fact that he made practically no changes in clinical and otolaryngological phases of this subject in the years intervening between the publication of the two volumes is a striking commentary on the accuracy of his observations and his faith in his own theories.

CARLETON BUILDING.

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XVI.

THE FIFTH, NINTH AND TENTH NERVES IN BRONCHIAL ASTHMA.*

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Every unbiased practitioner of medicine is familiar with the various outbursts of enthusiasm over some new cure for bronchial asthma during the last forty years.

I begin this discussion by offering an opinion which, upon first thought, will be branded by many as being extremely dogmatic. This opinion is that, up to the present time, bronchial asthma, as a problem, has had and still has the profession, as a whole, in a state of confusion. The claim is based upon personal experience covering a rather extensive clinical and experimental research, together with comparative analysis of the writings of such qualified students as Rackeman,[1] Tobey,[2] Kern and Schenck,[3] Shea,[4] Francis,[5] Ramirez,[6] Bray,[7] Adam,[8] Beckman,[9] Cameron,[10] Myers,[11] Haseltine,[12] Garretson,[13] Alexander[14] and others, and it is further confirmed by personal interview with large numbers of patients suffering from the condition, and the general practitioners throughout the various areas of the United States, both of whom the writer has had ample opportunity of contacting in the last seven years.

This state of affairs, together with being located in an area where these sufferers are sent by the hundreds every year from all parts of the country, has been chiefly responsible for stimulating this investigation, which now covers over 1,500 consecutive cases, including a wide variation in age, sex, type and duration.

During this program of detailed observation and the tabulation of events, a group of very significant and interesting facts came to light. In over 92 per cent of the entire group the attacks were initiated, became worse and reached the peak of severity between midnight and morning. Extrinsic conditions, such as sudden lowering of atmospheric temperature, cold waves, increased humidity, pollens, dust or contaminated air, which will produce nasal congestion in all

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noses, would incite or aggravate bronchospasm in over 87 per cent of these patients. In 72 per cent, bronchospasm in modified and mild form could be artificially produced by the mechanical stimulation of the ethmoid area in the nose, Arnold's nerve in the external auditory canal, especially in the presence of impacted cerumen, or stimulation of the retina by a constant beam of powerful light. In this particular group nearly 90 per cent manifested this susceptible area to be located in the ethmoid area of the nose, and complete anesthetization of this area will abolish the spasm previously induced. This can be accomplished with cocain or several other drugs and so is not a specific reaction to the solution. The remaining 10 per cent were divided between the mouth, throat, ear canal and the eyes. In the females, 27 per cent manifest their attacks to be definitely related to their menstrual cycles, and 5 per cent of the males were week end repeaters. Laborers who were spasm-free all week while working hard, perspiring freely, drinking an abundance of water and eliminating. With the whistle on Saturday noon they retired into a state of feasting and relaxation only to awaken after midnight Sunday in bronchospasm, which continued until their return to labor, when it was relieved spontaneously.

These and subsidiary factors, too numerous to mention, must lead one to suspect that in bronchial asthma there are two primary underlying mechanisms: one a generalized systemic or metabolic upset. Under this load the asthmatic staggers 365 days out of the year and assumes the potential position of a loaded gun. Superimposed upon this is a fluctuating or trigger mechanism which fires the shot, and the attack is on.

Space will not permit even mentioning the evidence which has been uncovered to support the metabolic abnormality side of the question except to refer to Table I which presents, at a glance, the lack of balance in physiologic constants in ten cases and to refer the reader to work already published,[12 8 10 15 16]. The attempt and purpose of this discussion is to present the findings with relation to the trigger mechanism because, as will be seen, the responsibility for its management apparently falls into the arms of the specialist dealing with the head and upper respiratory tract.

Since such a large percentage of the clinical patients manifested evidence of a trigger area in the combined regions of the eye, ear, nose and throat, it was decided to expose dogs to crucial experiment with proper connections between their bronchioles and recording devices and note the peripheral effect from electrical (Faradic current) stimulation of these areas. The detailed report of these experiments,

TABLE I.

Patient	Avg. Daily Oral Temp. Degrees	B M R	B P	Mg. Blood Sugar N—100 Mg.	Gas. Acid F.	Gas. Acid Tot.	Deg. Acid N—40°	Urine 24 hours N—25 G. Sp. Gr.	Urine 24 hours N—25 G. Urea Grams	Urine 24 hours N—25 G. Indican	Skin Appearance
H.M.—25—Male	97.6	85/50	—12	87	0	15	90	1026	12	++	Dry, scaly, sallow
P.M.—35—Female	96.8	90/60	—10	90	5	12	65	1015	14	+	Dry, icteric, thick
J.W.—37—Male	96.6	87/40	—15	85	0	10	110	1030	8	++	Dry, eczematoid
M.M.—48—Female	97 8	108/70	—12	87	0	6	102	1020	10	++++	Dry, thickened, scaly
M.G —54—Female	96	102/70	—18	85	5	15	90	1025	12	++	Dry, sallow, thick
M B.—29—Female	97	98/60	— 6	92	10	18	62	1024	14	+	Slight moisture
D B.—7—Male	98	90/55	+ 2	85			102	1018	8	+	Dry, eczematoid
G.S.—8—Female	98	90/60	+ 2	87			64	1010	8	+	Dry, eczematoid
M.N.—10—Male	96.8	85/50	—10	85			72	1008	12	0	Dry, scaly
B.I.—16—Male	97	88/55	—12	87	2	12	80	1016	10	0	Dry, eczematoid

Illustrating in ten cases of bronchial asthma the evidence of systemic upset as shown by a deviation from normal in various physiological constants. Note the constant changes in urine acidity, urea and indican. Also the abnormal skin and blood sugar levels.

together with the physiology involved, has been reported in connection with a London Medical Journal Symposium.[17] Reference to Fig. 1 will reveal the apparatus by which, through a small tracheotomy opening, the smaller group of bronchioles can be cannulated and their contraction and expansion recorded under the various extrinsic manipulations. It will be seen in Fig. 1 (a) that when the vagus is isolated, divided and the peripheral end stimulated a marked and definite bronchiolar constriction ensues. Stimulation of the central end, Fig. 1 (b), with a weak current likewise produces a constriction with inhibition of respiration. In this instance the impulses must travel through the center and down the opposite side. Stimulation of Arnold's nerve in the ear canal, Fig. 1 (c), produces a less marked but nevertheless a definite, bronchial constriction. In this procedure we have established a definite neuro-anatomic connection between the vagus and the bronchioles since, if we now cut both vagi (C1), all the above reflexes are abolished. Fig. 1 (d) represents stimulation of the central end of the sectioned superior laryngeal nerve (branch of the ninth). This produces coughing and marked inhibition to respiration. In Fig. 1 (e) bronchoconstriction was produced by injection of pilocarpine, and now, interesting to note, stimulation of the peripheral cut vagus produces a bronchial dilatation. Atropin now injected abolishes both reflexes. Pilocarpin is a stimulant to the vagal endings, increasing the tonus of peripheral structures innervated by it. Electrical stimulation is now apparently more effective to the inhibitory fibers, thereby producing relaxation. This phenomena has been demonstrated vividly by Dr. A. J. Carlson in his study of reflex action connected with various viscera. Atropin paralyzes the parasympathetic endings and thereby abolishes the pathway for the impulses. Stimulation of the ethmoid turbinal of the nose, Fig. 2 (a), with both vagi intact, produces a broncho-constriction as marked as in Fig. 1 (a). The section of both vagi or administration of atropin (A^1) will obolish the reflex completely. This train of events demonstrates not only the presence of a reflex hook-up between the nasal cavity, ear canals, mouth and throat and the bronchioles, but they indicate that the pathway is via the vagus. That this reflex hook-up is not general for all sensory nerves is indicated in Fig. 2 (b), where stimulation of the central end of the divided greater sciatic nerve produces increase in respiration but no bronchial constriction.

The clinical significance of these experiments in bronchial asthma becomes self evident, but it should be emphasized at this point, the many cases of prolonged cough which the internist is unable to elimi-

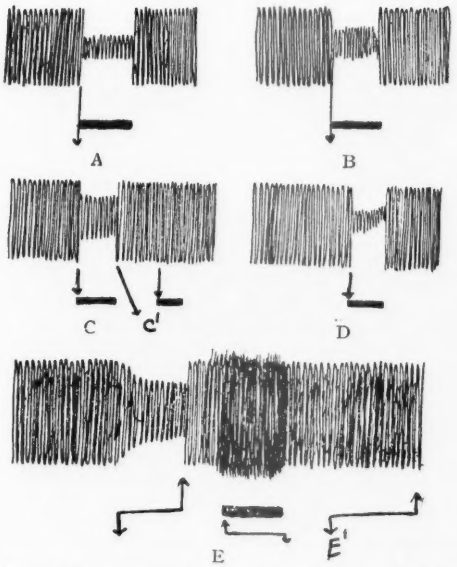

Fig. 1. *A* Stimulation peripheral end of cut vagus weak induction currents. *B* Stimulation central end of cut right vagus. *C* Stimulation sensory terminals in external auditory meatus (Arnold's nerve). C^1 Section of both vagi abolishes the reflex. *D* Stimulation central end of cut right superior laryngeal (branch of ninth) nerve. Cough is produced and marked inhibition to respiration. *E* Bronchoconstriction produced by pilocarpine. Stimulation of peripheral vagus now produces bronchodilatation. E^1 Atropine abolishes the reflex.

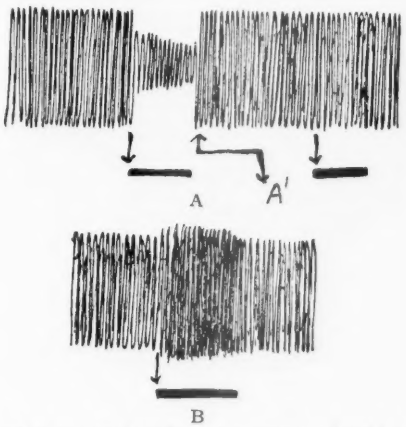

Fig. 2. *A* Stimulation (weak induction current) ethmoid turbinal with both vagi intact. A^1 Section of both vagi abolishes the reflex. *B* Stimulation central and cut right greater sciatic producing increase in respiration but no bronchoconstriction.

nate, due to the lack of consultation with the rhinologist, laryngologist or otologist.

A campaign of thought was now entered upon with the aim of attempting to combine these findings with the other side of the question, so evident in the asthmatic patient, namely, a generalized systemic or metabolic abnormality. Should we succeed in producing the two combined in a living animal we might possibly be able to produce bronchospasm in such an animal. In previous studies[18 19] we had succeeded in producing systemic toxicosis in living animals by the surgical production of isolated intestinal loops (see Fig. 5). The degree of toxicosis can be measured by the blood chloride and nonprotein nitrogen curves. We had likewise succeeded in producing endocrine upsets with tetany by total extirpation of the parathyroid glands.

A series of dogs were thus prepared, allowed to recover and at various succeeding intervals secondary procedures carried out with respect to the nasal cavity. The results are illustrated in Table II and are self explanatory. The detailed discussion beyond the scope of this report is given in the publication[17] above referred to. These animals have been demonstrated to many of our colleagues, and veterinarian consultation has confirmed that the picture represents animal bronchial asthma.

GENERAL DISCUSSION.

It is evident from both clinical and animal experimental work that there exists a neuro-anatomic reflex connection between the bronchioles and the sensory terminals of the fifth, ninth and tenth cranial nerves. Since both atropin and section of the vagi in the animals will abolish the reflex it would seem that the tenth nerve is the efferent pathway. However, in relation to the problem of bronchospasm, it is important to recall that stimulation of the cut cantral vagus would produce the same reflex through the opposite side. This finding so clearly explains the emphatic gospel preached throughout the writings of Chevalier Jackson,[20] Clerf[21] and others regarding the significance of foreign bodies in the bronchi, mediastinal growths, lung tumors, etc., in bronchial asthma. The vagus trunk carries afferent as well as efferent fibers, and the above conditions can and do undoubtedly act as trigger areas. Their presence must always be ruled out. La Forge[22] has written much in regard to his finding unilateral or ipsolateral bronchospasm—the spasm occurring on the side corresponding to the nasal pathology. These reflex experiments explain it and we have seen many clinical cases in our group in whom

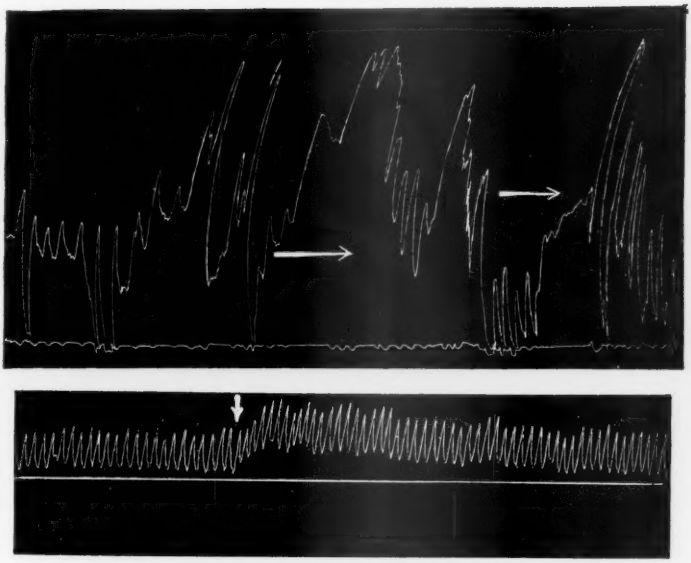

Fig. 3. Tracing resulting from mechanical and chemical stimulation of ethmoid turbinal in nose of laryngectomized patients in whom the bronchus was cannulated (after Professor Sercer, Zagreb, Jugoslavia).

we found it, and some in whom we have been able to produce it by stimulation of that side of the nose.

At this point any physiologic minded clinician will raise the question as to why do we obtain spectacular results by proper eradication of nasal pathology in some asthmatics and then see in succession a hundred patients with identical pathology who never would manifest bronchospasm? Our work attempts to answer this in two ways. First of all it indicates that in the asthmatic a systemic metabolic upset is present. Amongst other things which this might manifest is probably a change in nervous threshold which now renders the reflex centers more susceptible to afferent stimulation and they act by bombardment.

This is first indicated by the fact that mechanical or chemical stimulation of these trigger areas in the nonasthmatic will not produce any evident bronchospasm, although we have, upon many occasions, seen it produce coughing and sneezing, indicating the presence

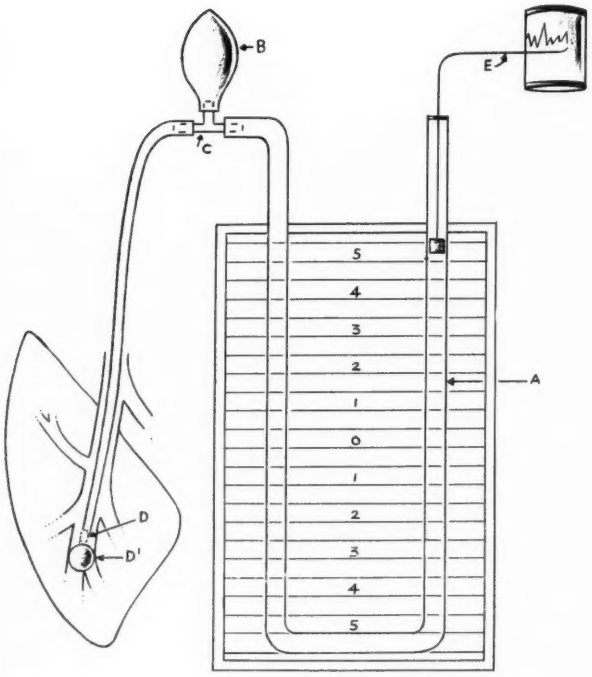

Fig. 4. Schematic illustration of manometer, canulization and recording apparatus. *A* Water manometer. *B* Inflation bag. *C* "T" tube. *D* Balloon collapsed and tucked inside tube. D^1 Balloon inflated after cannulating bronchus. *E* Recording pointer.

of the reflex. Secondly, a thorough physical, metabolic and biochemical study by a competent internist will invariably reveal abundant evidence of such upset. A thorough analysis of the cases in whom eradication of nose or throat pathology entirely alleviated the bronchospasm has, in our experience, invariably revealed that it was a combined pathology capable of producing both a systemic toxicosis and serving as a reflex trigger at the same time. Dr. Francis Wiley, in Boston at present, is emphasizing that he is finding microscopic multiple abscesses in the linings of antræ in which clinical and x-ray evidence are lacking in showing them to be pathologic. What else could be expected but that such a focus could serve a double function. Sinus irritations and infections, polyposis, septal deviations and

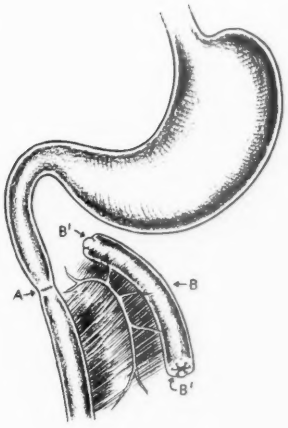

Fig. 5. Diagram illustrating production of isolated intestinal loops. *A* End to end anastamosis following removal of segment of jejunum. *B* Isolated intestinal loop with blood supply intact. *B*[1] Inverted and purse stringed ends.

spurs with mechanical impingements become so important that we have never yet seen a cure of bronchial asthma in a mouth breather. Tonsils and adenoids speak for themselves as well as all mouth deviations and impactions. I must caution you regarding impacted cerumen, suppurating chronic ears and mastoids. Look for them in every case. You will see spasm incited time and time again while you are irritating Arnold's nerve during removal of wax and it will at times last as long as twenty-four hours if you have been rough and produced abrasions. That these reflex pathways do exist has been confirmed by Brodie and Dixon[23] in crucial animals and in clinical patients following laryngectomy by Professor Sercer[24] of Zagreb, Jugoslavia. Reference to Fig. 3 reveals bronchial reflexes under the same mechanisms of production as in the animals.

It must be remembered that these asthmatic people are hyperaesthetic individuals and the rhinologist, otologist and laryngologist's work and responsibility on the whole deals with the very trigger area of their attacks. Super technique is automatically demanded. The rhinologist whose only resource is mutilating surgery in these cases is just as helpless as the internist whose only hope is serums and vaccines. Both are extremists of a different type, both have done harm, and both are responsible for the present terrible pessimistic attitude among

TABLE II.—PROTOCOLS OF ANIMALS.

Dog	Primary operation	24 to 52 hours	Second operation	24 to 48 hours	Third operation	Remarks
12	Intestinal loop	Loop distended; dog toxic; dehydration; chlorides low	Wire rings sutured in ethmoids	Thirty-six hours sneezing; watery nasal discharge; mild dyspnœa; crackling râles	Removal of loop and rings on sixth day	Prompt recovery; died in accident 3 months later
Spot	"	Loop distended; vomiting; dehydration; chlorides low	"	Thirty hours sneezing and secretion; 48 hours dyspnœa with moist râles; slight wheezing	"	Recovery normal until died of distemper 3 months later
14	"	Listless; loop distended; toxic; falling chlorides	Infected gauze to both antræ and ethmoids	Listless; vomiting; dehydrated; purulent nasal discharge; wheezing, cooing râles	Pilocarpine	Salivation; vomiting; diarrhœa; marked dyspnœa; death in 10 minutes; atropine, no effect; autopsy; loop intact; nasal suppuration; no pneumonia
Red	"	Toxic; increase N. P N.; low chlorides	"	Extreme toxæmia; glucose and saline by vein; marked wheezing, profuse râles	Pilocarpine	Increased dyspnœa; vomiting, sweating, diarrhœa; atropine abolished; died 12 hours later. Autopsy; no pneumonia; loops intact
16	"	"	"	Loud wheezing upon exertion or excitement	Pilocarpine	Salivation; sweating; severe dyspnœa; atropine abolished; died, 18 hours. Autopsy; purunose: no pneumonia
Blacky	Infected gauze to antræ and ethmoids	Sneezing; nasal discharge		Although dyspnœa, no evidence of cooing râles		Dyspnœa on fifth day; died on seventh day. Autopsy; aspiration pneumonia
19	Rem. parathyroids	Tetany; controlled with Ringer's solution	Inf. gauze to nose	Toxic; wheezing; diffuse râles, cooing; and moist muscles spastic		Died third day in tetany. Autopsy; supp. ethmoiditis; lungs free from pneumonia; hemorrhages, intestinal mycosa
20	"	"	Glycerine injected into turbinals on both sides	Nasal block; watery secretion; sneezing	Decomposed meat given on tenth day	Extreme tetany with death. Autopsy; œdema, nasal tissues; petechial hemorrhages intestine, normal lungs

both doctors and laymen. The writings of Haseltine[12 25 26 27] are most valuable and instructive in this regard.

If this work indicates anything of clinical value, it certainly indicates that cooperative team work between the rhino-oto-laryngologist and internist in every case is necessary if the proper attack to the problem is to be accomplished. When this demand is satisfied it has been our experience that the clinical results justify both the theory and the demand.

CONCLUSIONS.

1. That two mechanisms are at work in the production of bronchospasm, namely, a generalized metabolic abnormality constantly present and a superimposed trigger mechanism which fires the attacks.

2. Evidence is produced to indicate that the trigger mechanism works through neuro-anatomic reflex pathways.

3. Experimental bronchospasm has been produced in both the crucial and living experimental dog and in the human being by utilizing these pathways.

4. Evidence is produced to point out various factors entering into the metabolic upset.

5. A working cooperation between the ear, nose and throat specialist and the internist is necessary in all cases in order to properly combat the problem.

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XVII.

THE AUTONOMIC NERVOUS SYSTEM: SOME FUNDAMENTALS FOR OTOLARYNGOLOGISTS.*

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SAN DIEGO.

An adequate conception of the physiology of the autonomic nervous system should be based on anatomy and terminology, both of which have been made unduly difficult. The advantages have not been stressed as much as the difficulties and this has resulted in the present obscurity in reference to this system.

Although we frequently encounter statements which lead us to think of the autonomic system as separate and independent, we must bear in mind that actually there is only one nervous system for the entire body. This system is completely interrelated, but its functions are distributed along two sharply contrasting lines and are facilitated by a corresponding anatomic separation. One is called the cerebrospinal or sensori-motor system because it has to do with sensations and reflex motions which are typically under the control of the will, rise to the level of consciousness, have representation in both the cerebral cortex and cerebellum, and they exist for the purpose of satisfying the essential needs registered by the autonomic system and the maintenance of the body in relation to external environment.

The other is called the autonomic nervous system. It is a system which is related to the structures of the body which function automatically, and, phylogenetically, is the outgrowth of those primitive networks which constitute the sole nervous system of unicellular organisms. This conception attaches such fundamental importance to the autonomic system that we may think of it as touching in upon every structure of the body, either stimulating or inhibiting actions to best serve its general economy. It regulates the glands of external and internal secretion, the size of blood vessels, the action of smooth muscle, is concerned with immunity and sensitivity, enters into the emotional life of the individual and generally serves to maintain the normal state of the body. Its activites are below the level of consciousness and are not under control of the will.

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Both of these are reflex systems characterized by the typical three neurone arc which is made up of a receptor, connector and effector neurone. The receptor carries sensation centrally into the cord, where a connection is established with a second neurone which contacts the effector, or third neurone. Both of these systems are designed and exist for the sake of the third or effector neurone and have no function except response to stimulation.

In the development of the embryo at the stage of six millimeters the neural canal shows evidence of a division into dorsal and ventral columns. This is accomplished by an enlargement of the canal opposite the center and a corresponding depression in the nervous tissue surrounding it. Thus there is established a constriction known as the sulcus limitans. On the dorsal side of this develop all the sensory nuclei of the brain stem and cord, and on the ventral side develop all the motor nuclei. This separation of motor and sensory columns continues throughout development. Mixed nerves show their origin by two roots, one in each segment, and pure motor and sensory nerves show their roots developing well within the limits of their respective columns.

It is important for our concept of the autonomic system to note that it shows an early line of departure from the cerebrospinal system by a migration of its cells out of the neural canal along the anterior or motor roots and that they proceed to their destinations along quite different pathways. Here is a suggestion that the particular purpose may be to insulate it against interference by the activities of the central nervous system, which are volitional motor responses to conscious sensations.

The autonomic nervous system is separated into two main divisions, each of which has a function distinct from the other and a different anatomical arrangement of its neurones. These are known as the sympathetic and parasympathetic.

The sympathetic system is characterized anatomically by a chain of ganglia which is close to the spinal cord. Each ganglion is connected with its corresponding segment of the cord. This distribution holds throughout the entire thoraco-lumbar area. The first or receptor neurone has its nutrient cell in the dorsal spinal ganglion and enters the cord through the posterior root, along with the afferents of the sensori-motor system, and terminates in the lateral horn of gray matter. The connector or second neurone has its nutrient cell in the lateral horn of gray matter of the cord. It passes out the anterior roots and joins the mixed spinal nerve for a short distance and then enters its sympathetic ganglion.

Because it is myelinated it is frequently called the white ramus communicans, or preganglionic neurone. After making a synapse in the ganglion the third or effector neurone re-enters the mixed spinal nerve to reach its ultimate destination. The effector neurone is not myelinated and it is frequently termed the gray ramus communicans or the postganglionic neurone. A succession of these ganglia in close proximity looks like a chain of beads and is often called the ganglionated cord. The relationship for the cervical spine is different. For the eight cervical nerves there are three ganglia, known as the superior, middle and inferior. The first four nerves are connected with the superior, the fifth and sixth with the middle, and the seventh and eighth with the inferior. This is the classical description and will suffice to fix the arrangement in our minds, although actually the middle ganglion is sometimes absent and the first thoracic ganglion is frequently fused with the inferior cervical to form the stellate ganglion. Postganglionic fibers are distributed to the third, fourth, fifth, sixth, ninth, tenth and twelfth cranial nerves. The superior ganglion supplies the ninth, tenth and twelfth directly and the others by fibers conveyed along the internal carotid artery and its branches.

The parasympathetic system is characterized by ganglia which are located at great distances from the cord, in or near the tissues which they supply. The afferent fibers are located above the cord at the level of the midbrain and medulla. They contact their respective effector neurones and peripheral ganglia by a distribution through the third, seventh, ninth and tenth cranial nerves. Because of the greater importance and extensive distribution of the tenth nerve this system is sometimes referred to as the extended vagus system.

The final distribution of both sympathetic and parasympathetic effector neurones is in identical tissue, thus creating a double autonomic innervation with generally antagonistic effect. Thus the sympathetic dilates the pupil and the parasympathetic contracts it. There is, however, one exception; both of them increase the action of lacrimal and salivary glands.

We should now have clearly in mind that the cervical ganglia are the cell stations for all the autonomic fibers which innervate structures in the head from the thoracolumbar or sympathetic system, and that the cranial autonomic or parasympathetic fibers have their cell stations in four great distal ganglia, viz.: the ciliary, otic, submaxillary and sphenopalatine.

The Origin and Function of the Parasympathetic System.—The cranial parasympathetic is made up of connector or preganglionic

fibers which leave the brain by the third, seventh, ninth, tenth and eleventh nerves. The third, seventh and ninth terminate in cranial ganglia, viz.: the ciliary, sphenopalatine, otic and submaxillary. The fibers which make up the tenth and eleventh are not not considered in this paper.

The Third Nerve.—The function is contraction of the pupil when light falls upon the retina. Connector fibers arise from the Edinger-Westphal nucleus in the floor of the aqueduct of Sylvius and travel with the oculomotor nerve to reach the ciliary ganglion. A synapse is made in the ganglion, and postganglionic fibers enter the constrictor muscle of the iris.

The Seventh Nerve.—Preganglionic fibers take origin in the superior salivary nucleus. They pass by the nerve of Wrisberg or nervus intermedius, through the geniculate ganglion by way of the great superficial petrosal nerve, to the sphenopalatine ganglion, where a synapse is made. Postganglionic fibers pass with the maxillary branch of the fifth nerve to the lacrimal gland by way of the zygomatic nerve and to the mucosa of the nose and palate. Other fibers continue in the facial nerve and, subsequently through the chorda tympani, join the lingual nerve to reach the submaxillary ganglion. Postganglionic fibers enter the submaxillary gland. The function is to increase secretion.

The Ninth Nerve.—Preganglionic fibers connected with the ninth nerve arise from the inferior salivary nucleus. They reach the otic ganglion by way of the tympanic and small superficial petrosal nerves. Post ganglionic fibers go to the buccal mucosa and the parotid gland in the auriculotemporal nerve. The function is to increase secretion. Other preganglionic fibers contact small ganglia in the area of the tongue supplied by this nerve.

The Origin and Function of the Sympathetic System.—The internal carotid plexus gives rise to the internal and external carotid nerves. The internal carotid nerves adhere in the internal carotid artery and become separated finally to form the cavernous and internal carotid plexuses. The latter sends branches to the abducens nerve and gasserian ganglion. It gives rise to the deep petrosal and caroticotympanic nerves. The deep petrosal joins the great superficial petrosal to form the vidian or nerve of the pterygoid canal and passes through the sphenopalatine ganglion to terminate as vasoconstrictor and secretory fibers in the posterior nasal nerve.

The cavernous plexus sends out filaments to the oculomotor, trochlear and ophthalmic nerves, and also through or around the ciliary ganglion. It also establishes a connection with the hypophysis.

The tympanic plexus is situated in the medial wall of the middle ear and supplies the mucosa of the tympanum, mastoid cells and eustachian tube.

The peripheral autonomic plexuses then are made up of short postganglionic fibers from the parasympathetic ganglia and also the long postganglionic fibers of the sympathetic system which terminate in identical tissue. Regardless of the particular route by which fibers arrive at their destination they remain in functional relation with their nuclei of origin. The cranial parasympathetics are distributed along the branches of the fifth nerve by four great ganglia—ciliary, sphenopalatine, otic and submaxillary—but are in no more functional relation to it than are the nerves in the cavernous sinus to the venous circulation.

Out of such facts as have been briefly referred to, certain general conceptions of the functional significance of these two divisions of the autonomic system have been created. The parasympathetic is regarded as one which has to do with anabolism. It is protective, conservative and acts constantly even during sleep. It contracts the pupil, retracts the eyeball, produces a flow of gastric and salivary secretion, maintains the tone of the intestinal canal, influences absorption and elimination, and slows the heart. The sympathetic division is the emergency mechanism and the catabolic factor in metabolism which sustains us under stress. It dilates the pupil, protrudes the eyeball, responds to pain, hemorrhage, shock, emotional strain, temperature, fatigue, and prepares the body for both attack and defense.

We see in most individuals a well balanced antagonism between these two systems, but frequently we may also observe cases in which one or the other system shows a dominant effect. The terms vagotonia and sympathicotonia have been applied to these conditions. This idea, first advanced by Eppinger and Hess, has lacked sufficient support in the realm of therapy and clearly defined symptomatology to warrant stress, but we must not lose the grain of truth contained in it. For example, the individual who gives a history of asthma, has a low blood pressure, slow heart, cold extremities, may be considered as living under overactive parasympathetic or vagus stimulation, and we also find in support of this that he has tolerance for adrenalin. An example of sympathicotonia is seen in the individual who has dilated pupils, excessive sweating, elevated blood pressure, flushed skin, warm extremities, and who is excessively stimulated by adrenalin.

Clinically there are definite conditions which are due to the predominance of the sympathetic or thoracolumbar system. In general

such disturbances as Raynaud's disease, high blood pressure, intermittent claudication, Hirschsprung's disease, and globus hystericus, are examples of vaso- and viscero-spastic states which can be relieved by sympathectomy but which may some day yield equally well to a therapy which will antagonize overaction of the sympathetics.

Any discussion of the autonomic nervous system before a body of otolaryngologists must bring to mind the issue of the sphenopalatine ganglion in its relation to pain. It belongs to the parasympathetic system and is a component of the seventh nerve. It is a cell station where a synapse is made for postganglionic fibers passing to the lacrimal gland by way of the zygomatic nerve and to unstriped muscle and blood vessels and glands of the palate and nasopharynx via the palatine nerves. The preganglionic fibers are called the great superficial petrosal nerve. This nerve travels through the Vidian canal before reaching the ganglion, and here the great deep petrosal nerve of the sympathetic system comes into relation with it by reason of traveling through the same canal. Together they form the vidian nerve or nerve of the pterygoid canal. The deep petrosal passes through or around the ganglion on its way to excite sweat glands and exercise vasoconstrictor effects on blood vessels.

The terms sphenopalatine ganglion neuralgia and vidian neuralgia are firmly intrenched in our literature owing to the sincere and convincing manner in which Sluder and Vail have expressed their views. The question raised is whether or not they will withstand the closest scrutiny. The great mechanism for conscious sensation in the head is the fifth nerve. Its ophthalmic and maxillary divisions innervate the whole nasal mucosa, including the sinuses. To reach the nasal mucosa the maxillary nerve sends fibers down to the sphenopalatine ganglion to accompany the autonomic fibers through the sphenopalatine foramen. Anatomists and physiologists are agreed that there is no peripheral functional relationship between the fifth nerve and the autonomic system. The whole philosophy of sphenopalatine ganglion neuralgia is based upon the conception that pain is a function of this autonomic ganglion. It is fortified by the fact that so-called cocainization of the ganglion relieves pain. However, the fibers of these nerves are in such close anatomic relationship that it is impossible to cocainize, inject or surgically remove the ganglion without equally affecting the fibers of the maxillary nerve.

It becomes immediately apparent then that no phenomena can be ascribed solely to the effect upon the ganglion when any of these procedures are carried out. In support of this statement we have no reports of cases which showed the syndrome of pure autonomic

paralysis after injection or cocainization, and no cases which showed pure autonomic stimulation as evidence of disturbance in the ganglion.

A great physiologist, Sherrington, has definitely established the law of forward progression. It means that a nerve impulse can cross a synapse only in the direction of its functional reflex. This is a fundamental fact of such importance that it should eliminate the term antidromic impulse. By this term authors carry us at will forward and backward along the nerve pathway and build up a totally untenable hypothesis. They would have us believe that the same nerve will transmit efferent impulses to induce vasoconstriction and glandular secretion and also afferent impulses for conscious sensation.

The results of section of the sensory root of the fifth nerve, as seen after the operation for tic douloureux, are almost conclusive in themselves. After this procedure it is impossible to elicit pain by traumatizing the nasal mucosa, although the sphenopalatine ganglion and the vidian nerve are still intact.

It seems logical to ask this question: If the autonomic system transmits sensations of pain, what role shall we attribute to the fifth nerve, or conversely, how will we explain the absence of pain in the broad expanses of the body cavities which are so plentifully supplied with fibers of the autonomic system?

One is confused by the great variety of symptoms and diffuse distribution of sensations which have been attributed to sphenopalatine ganglion neuralgia. It has become the vogue to make the autonomic nervous system a great stamping ground for unknown causes and unexplained symptoms. Quite aside from the pain which by distribution can be assigned to a particular nerve, there are certain atypical neuralgias which are entirely irregular and are not relieved by section of the sensory root of the fifth or by removal of foci of infection.

There is one other route by which sensation in the head may reach the sensorium, viz.: through the sensory or geniculate ganglion of the seventh nerve. The function of this structure, however, seems to be limited to deep pressure sensibility and the severe neuralgias are never attributed to it. Such painful sensations in the head as occur after section of the fifth nerve can be better understood as psychalgias and neurosis. A considerable number are allergic, many show sinus infection and some are neurotic. The latter class is perhaps the least understood. In my experience, many have been women who had little responsibility, ample means, and no vital interests. Under such cir-

cumstances, there are few impediments to development of a functional neuroses. In such cases one can understand the failure of opiates to relieve pain. The resistance to these drugs in neurotic and hysterical states is well known. It explains the relief which has been obtained in a few cases by injection of normal saline instead of escharotics, for the technic is equally suggestive of a formidable therapeutic procedure.

I have found fixed convictions and subterfuge to be the real basis of the complaint which lead to a diagnosis of sphenopalatine ganglion neuralgia in a few cases. In these cases an investigation and understanding of the emotional factors involved was the greatest aid in determining the true cause. Two cases may be reviewed as illustrations. One, a secretary, who for two years had complained of severe pain which was diagnosed as sphenopalatine ganglion neuralgia. She had received a great deal of treatment with increasingly frequent applications of cocain and two ganglion injections. One injection did no good and the other gave some relief. Finally she was told by a long suffering employer that she must either get something done to eliminate the trouble or she would be replaced. After that cocainization helped definitely. She asked for another injection, which gave permanent relief. She had reached a crisis in which playing upon the sympathies of her employer no longer accomplished the desired effect and with the latest injection and dire necessity as a vehicle she transported herself out of a fixed conviction. Another case is a woman with no responsibilities who became convinced that in her evening bridge club she had discovered a contender for the affections of her husband. She began soon to complain of severe pain in her head. When I first saw her she described the pain as being present at the time and unbearable in severity, and requested treatment. She had received regular cocainizations for eighteen months. There was something about her manner and statements which was not convincing. I got from her the statement that she was desperately afraid of losing her husband and asked that I inform him that the night air was particularly bad for her trouble. By such subterfuge she was trying to prevent a domestic crisis.

In this brief review of the anatomy and physiology of the cranial autonomic nervous system some statements are made contrary to the prevailing opinions. I have ventured to differ from them by selecting certain known facts in anatomy and physiology which seem to give a clearer, more logical, and more probable explanation of the pain which has hitherto been attributed to the autonomic system. This concept is in keeping with the idea of a fundamental division of the

nervous system into two functional systems, viz.: sensorimotor and autonomic, in which each has its definite role. It attributes pain to the sensory division, viz.: the fifth, seventh, ninth and tenth nerves, which are known to have receptors for that sensation, and it leaves uncompromised the logical conclusion that the autonomic is an efferent system, distributed to glands, smooth muscle and blood vessels, and is concerned only with vegetative functions.

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XVIII.

PATHWAYS INVOLVED IN PAINS OF NASAL AND PARANASAL ORIGIN REFERRED TO THE LOWER CERVICAL AND UPPER THORACIC SEGMENTS AND THE UPPER EXTREMITY.*

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ST. LOUIS.

The causal relationship between lesions in the orbit and the mucous membranes of the nose and paranasal sinuses and certain pains in the neck, shoulder, arm, forearm and hand was first pointed out by Sluder. These pains exhibit the characteristic features of referred pains and, in some instances, are accompanied by other referred phenomena. For example, Terracol (1932) cited a case in which operation for the relief of nasal obstruction was followed by eruption on the chest and another in which drainage of the wound following a mastoid operation was followed by violent pains in the arm and eruptions on the chest.

According to the theory formulated by Head, the areas in which referred pains are localized always fall within segments of the body which are supplied by nerves which also send afferent fibers to the site of the causative lesion. This theory is supported by extensive data and probably is applicable in all cases of referred pain; consequently, pains in the neck, upper thorax and upper extremity associated with lesions in mucous membranes of the nose and paranasal sinuses cannot be regarded as referred from these lesions unless afferent spinal nerve components actually extend into the areas in which the lesions are located.

Afferent Spinal Nerve Components with Cephalic Distribution.—The earlier anatomic studies carried out to discover possible afferent spinal nerve components in the cephalic area have yielded only negative results mainly because it was assumed that such components, if present, would be incorporated in the cervical sympathetic trunk.

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From the Department of Micro-Anatomy, St. Louis University School of Medicine.

According to the best evidence available, the upper cervical portion of the sympathetic trunk includes no afferent fibers. Another pathway along which afferent spinal nerve fibers might extend into the cephalic region is provided by the plexuses on the common, internal and external carotid arteries. On the basis of an analysis of the clinical results of various surgical procedures carried out in the treatment of atypical facial neuralgia, Fay (1932) advanced the opinion that afferent components of the upper thoracic nerves traverse these plexuses and enter the area of distribution of the trigeminal nerve. This opinion has been confirmed by experimental anatomic findings (Kuntz, 1934). In experimental animals (cats), components of the upper thoracic nerves, which probably are exclusively afferent, have been found to traverse the inferior cervical sympathetic ganglion and join the plexus on the common carotid (Fig. 1) artery and extend upward in it. Most of these fibers continue cephalad in the internal and external carotid plexuses and probably reach their terminal distribution mainly through the terminal branches of the trigeminal nerve. The presence of myelin degeneration in Marchi preparations of nasal and nasociliary nerves following section of the roots of the upper four thoracic nerves, as observed by Christensen (1934), indicates that some of the afferent components of the thoracic nerves which extend into the cephalic region reach the mucous membranes of the nose and paranasal sinuses.

The demonstration of afferent components of the upper thoracic nerves among the afferent fibers which supply the mucous membranes of the nose and paranasal sinuses affords the anatomic basis for the interpretation of pains in the lower cervical and upper thoracic segments and the upper extremity associated with lesions in the nasal and paranasal mucous membranes as referred pains in conformity with Head's theory of localization.

Mechanism of Referred Pain.—The inadequacy of the older theories regarding the mechanism of referred pain has been pointed out repeatedly. Many clinical and experimental observations, furthermore, have contributed to a better understanding of referred pain and the phenomena associated with it. On the basis of extensive clinical observations, Wernoe (1925) advanced the opinion that referred pain associated with a visceral lesion has its origin in changes brought about in the skin through viscerocutaneous reflexes, i.e., the sensation of pain is the direct result of afferent impulses which actually arise at the periphery. In support of this opinion, he pointed out that under certain conditions sympathetic stimulation alone gives rise to pain. For example, exposure of the hands to low temperature

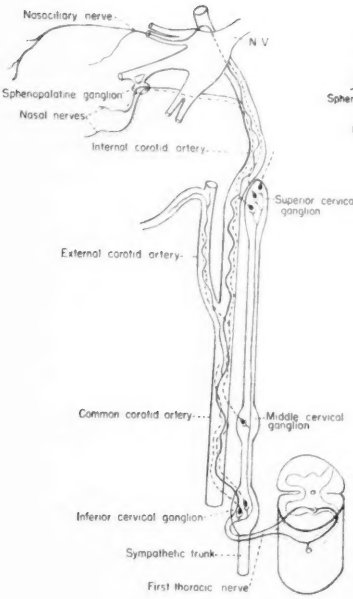

Fig. 1.

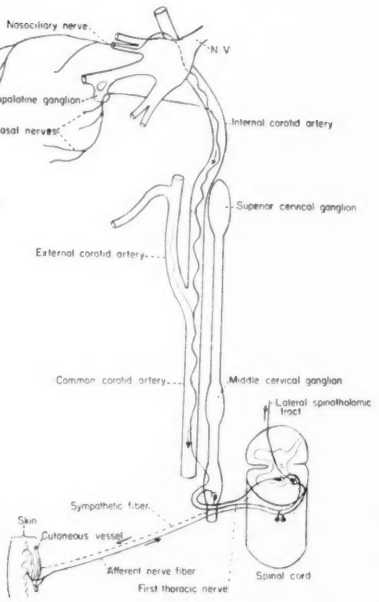

Fig. 2.

Fig. 1. Diagram illustrating the anatomical relationships of the spinal nerve components which extend cephalad in the plexuses on the common, internal and external carotid arteries.

Fig. 2. Diagram illustrating the probable pathways involved in pains of nasal and paranasal origin which are referred to the neck, upper thorax and upper extremity.

results in reflex vasoconstriction and ischemia, particularly in the fingers. As the ischemia becomes more marked, the fingers become painful until, with complete anemia, the anesthetic stage is reached. If the hand is then warmed the fingers again become painful until the ischemia subsides. Wernoe assumed that in like manner the cutaneous pain receptors may be stimulated by the ischemia brought about in the skin through viscerocutaneous reflexes elicited by the stimulating effect of a visceral lesion.

That the stimulation of pain receptors is a frequent accompaniment of peripheral vasoconstriction is well known, but there is no general agreement regarding the stimulating agent which acts upon these receptors. According to Lewis, Pickering and Rothschild

(1932), the pain associated with ischemic contraction of skeletal muscles results from a stimulus derived from the accumulation of metabolites in the tissues. The reported observation of Moore and Moore (1933) that the injection of a 0.1 per cent solution of lactic acid into an artery in an anesthetized animal strongly stimulates the afferent fibers which terminate in relation to the smaller arterial branches seems to support this assumption. Other investigators, particularly Popper (1933) and Schretzenmayer (1933), have advanced data which seem to support the view that chemical factors play a rôle in the causation of pain associated with ischemia. Reflex vasoconstriction resulting in ischemia in peripheral areas in which referred pains are localized is not uncommon.

The writer has previously emphasized the importance of reflex phenomena in the causation of referred pains (Kuntz, 1929, 1934) and has outlined the reflex mechanisms involved in these phenomena. They consist essentially of visceral or somatic afferent neurons through which afferent impulses are conducted from the site of the lesion or the irritable tissue adjacent to it into the cerebrospinal axis, visceral efferent neurons located in the intermediolateral cell column which conduct the impulses to the sympathetic trunk and sympathetic neurons located in the corresponding ganglia of the sympathetic trunk, through which efferent impulses are conducted to the smooth muscle in the area in which the referred pain is localized. One of the results of these reflexes probably is the stimulation of pain receptors located in this area by means of a chemical substance. The impulses arising in them, which result in sensations of pain, are conducted centralward through the ordinary pain conducting fibers. Pollock and Davis (1935) have outlined the reflex mechanisms involved in the causation of pain referred from the diaphragm in a similar manner. They also have supported the theory that the impulses which result in the sensation of pain localized in the area of reference actually arise in peripheral sense organs as a result of stimulation of these organs by means of an unknown mechanism.

Specific Mechanisms Involved in Pains Referred from the Nasal and Paranasal Region to the Neck and Upper Extremity.—According to the conception of the mechanisms of referred pain set forth above, the components of pains referred from lesions in the nasal and paranasal mucous membrane to the lower cervical and upper thoracic segments and the upper extremity, which are due to the reflex phenomena in the area of reference, can be explained most satisfactorily as follows: Impulses arising at the site of the lesion are conducted into the upper thoracic segments of the spinal cord through afferent spinal

nerve components which traverse the plexuses on the common and internal carotid arteries. In the spinal cord, these impulses are transmitted to preganglionic neurons in the intermediolateral column through reflex connections. These neurons conduct them into the corresponding ganglia of the sympathetic trunk where they are transmitted to sympathetic neurons. The latter, in turn, conduct them peripheralward. The reflex responses at the periphery include vasoconstriction, piloerection, sweat secretory activity, etc. These responses are accompanied by stimulation of peripheral pain receptors from which impulses are conducted into the spinal cord through afferent components of the corresponding spinal nerves and upward in the lateral spinothalamic tract on the contralateral side (Fig. 2).

SUMMARY.

Certain pains in the neck, upper thorax and upper extremity which are associated with lesions in the mucous membranes of the nose and paranasal sinuses exhibit the characteristic features of referred pains. In view of the anatomic data which indicate that afferent components of the upper thoracic spinal nerves traverse the plexuses on the common and internal carotid arteries and reach the mucous membranes of the nose and paranasal sinuses, these pains may be regarded as referred, in conformity with Head's theory of the localization of referred pains.

Referred pains are accompanied by reflex phenomena which play a part in the causation of the sensations of pain. Impulses directly responsible for sensations of pain probably arise in pain receptors in the peripheral area in which the pain is localized. The stimulation of these receptors is caused by the reflex responses, probably through the accumulation of waste metabolites or the liberation of a stimulating chemical substance.

The mechanisms involved in the components of the referred pains in question, which are due to reflex phenomena, may be outlined as follows: Impulses arising at the site of a lesion in the nasal or paranasal mucosa are conducted into the spinal cord through afferent components of the upper thoracic nerves, reach the corresponding ganglia of the sympathetic trunks through preganglionic neurons, and are conducted to the periphery through sympathetic neurons. Pain receptors at the periphery are stimulated; the impulses arising in them are conducted into the spinal cord through afferent components of the corresponding spinal nerves and upward through the lateral spinothalamic tract on the contralateral side.

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XIX.

CILIATED NASAL EPITHELIUM: ITS CULTURE IN VITRO. PRELIMINARY REPORT.*

ARTHUR W. PROETZ, M.D.
AND
MARIAN PFINGSTEN, A.B.

ST. LOUIS.

No matter how much may be gained in the differentiation of structure from a fixed and stained preparation, it is highly desirable to observe any organic tissue in the living state. When this tissue is essentially motile in its function, such observation becomes a necessity if one is to form a clear conception of its behavior. Just as a cadaver fails to suggest a proper picture of a man running, so a fixed preparation of ciliated epithelium does not reveal the mechanism of ciliary streaming.

Several years of experimental work with ciliated mammalian nasal epithelium, either removed from the animal and kept motile, or studied in situ, has stimulated us in the endeavor to grow such tissue. This is a preliminary report of the methods employed and the modest success attained.

The cultures grown have convinced us of the practicability of the method for the basic study of this tissue. It opens a gateway to the direct observation of epithelial regeneration, surface reactions in sensitized and desensitized individuals and innumerable other phenomena having to do with this all important first line defense against bacterial invasion.

The tissues employed were fragments of turbinates taken from the guinea pig fetus at term. This tissue is in no sense embryonic or immature (cilia can be detected as early as at 60 per cent gestation) but was chosen because of the facility of obtaining and implanting it without contamination.

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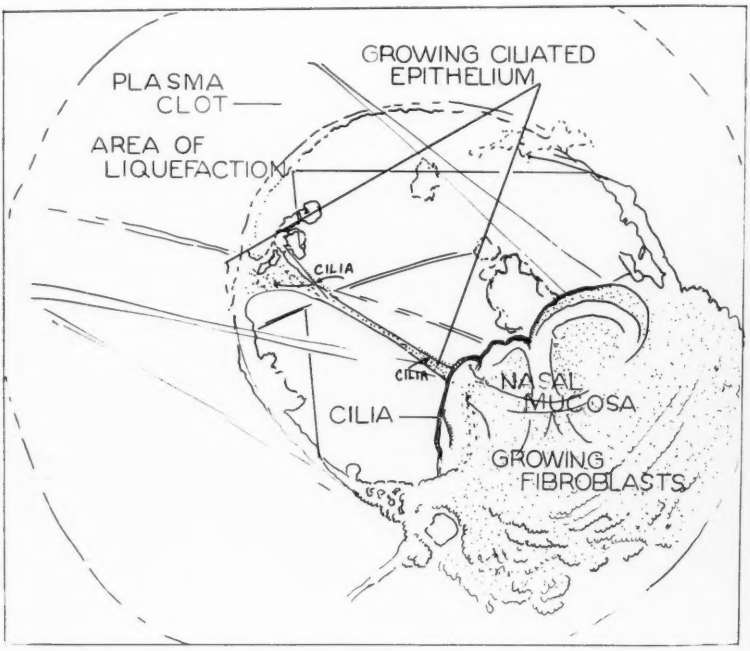
*From the Oscar Johnson Institute, Washington University School of Medicine. This investigation was made possible by a grant from the Chemical Foundation. Presented before the annual meeting of the American Laryngological, Rhinological and Otological Society, Denver, May 20, 1936.

The medium employed is essentially that of Vogelaar. It has been the especial aim of this investigator to determine accurately the significance of definitely known chemical substances and their effects upon tissue growth and differentiation. The feeding solution is one of chemically known composition which, when used with plasma and a calcium Ringer solution, has been quite adequate for the cultivation of both human and animal tissues. While we recognize the possible inadequacy of this medium for the best cultivation of epithelium, it seemed, to us at least, a likely point of departure in view of Vogelaar's observation that thyroid epithelium in his cultures showed an excellent growth along with that of the connective tissue. Human tissue grows well when the medium is made with either beef or sheep plasma. In our own experiments with the guinea pig, we have used plasma from the same animal, obtained at the time when the tissue was secured.

The culture medium is composed of equal parts of plasma, calcium Ringer solution and feeding solution, the latter exactly as described by Vogelaar. Omitting the details of preparation, for which the reader is referred to the original articles, this is a combination of magnesium Ringer solution, peptone, thyroxin, hemin, cysteine, glucose, insulin and sodium and potassium acid phosphates.

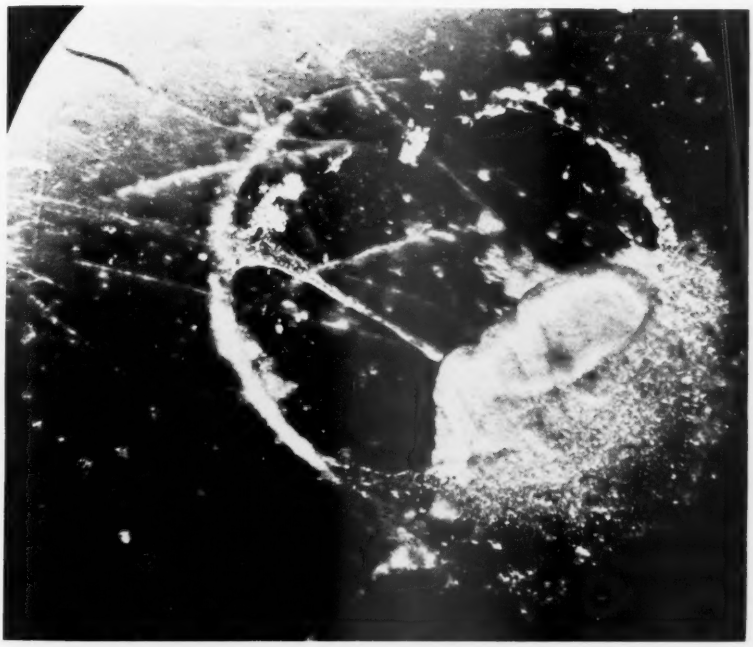
A pregnant guinea pig is bled to death directly from the heart under aseptic precautions in order to obtain blood for the plasma. The embryos are removed one by one from the incised uterus with a hemostat. The umbilical cord is cut and each animal is put into a sterile container with a small amount of sterile Locke solution or magnesium Ringer solution. The jars containing the embryos are placed in an aseptic glass cage in which all necessary sterile instruments have previously been prepared. The turbinates are obtained and minute pieces of epithelium from them are immediately transferred to the Locke solution, from which they are implanted as soon as possible in the medium. After the clots are formed the cultures are sealed with paraffin in a Maximow slide and cover slip and are incubated at 37.5° C. When liquefaction occurs the culture must be changed or refed.

This liquefaction of the medium is characteristic of the epithelial culture. In the accompanying cinemicrographs may be seen these zones of liquefaction always about the epithelial cells and not occurring on the opposite side of the implant where fibroblasts are multiplying. As the liquefaction progresses a strand of new epithelial cells can be seen to reach across it and to proliferate along its still coagulated wall. (Motion pictures of the culture showing the new epithelium accompanied the presentation.)



PHOTOMICROGRAPH OF CULTURE OF CILIATED EPITHELIUM.

The fragment of nasal mucosa is shown in the lower right corner, embedded in its clot of medium. The adjacent circular area is the liquefaction produced with the epithelial growth. The fibroblasts growing down and to the right show no



liquefaction. The strand of tissue extending up and to the left from the implant to the periphery is new epithelium covered with active cilia. Near the tip, and connected to a small mass of detached cells, can be seen a threadlike protoplasmic process, along which individual ciliated cells are found.

These strands of new epithelial cells are covered with active cilia whose characteristics of motion and speed of vibration cannot be distinguished from those of the mother cells. Sometimes only a portion of these new growths is ciliated. In one instance cilia were found upon the merest thread of new protoplasm, apparently the offshoot of a single cell. Ciliary streaming is quite apparent in the liquefied medium surrounding these cylindrical offshoots.

Beside their morphologic characteristics, the epithelial cells fortunately have a slightly yellowish tinge which differentiates them in the photographs from the fibroblasts. This contrast can be slightly augmented by the use of a pale blue filter (Wratten H, 45), but the slight advantage in contrast is more than offset by the loss of illumination.

The cinematography was done with a 16 mm. camera, single frames being exposed at twenty, forty or sixty second intervals. The shutter was tripped by means of an electro-magnet activated by a timing device especially constructed to supply instantaneous impulses at the required intervals.

Owing to the liquefaction which occurs, it is necessary to establish the incubator containing the microscope upon a vibration-proof foundation in order to prevent disturbance of the cells and jarring them out of the focal plane. The camera and all of its activating mechanism is suspended from the ceiling and just fails to make contact with the microscope beneath it, preventing any camera vibration from reaching the tissue.

* * * *

The authors disclaim any originality of method. The sole purpose of this communication is to report that ciliated epithelium with active cilia can be grown in such a manner as to permit of experimentation under direct microscopic scrutiny.

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XX.

SPONTANEOUS (NONTRAUMATIC) ATLANTO-AXIAL SUBLUXATION.*

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Otolaryngologists have a special concern in a type of subluxation of the atlas on the axis due to systemic infection.

Dislocation of the atlas on the axis, whether or not accompanied by rotation, is not a frequently reported condition. When it does occur, it is usually the result of violence.

There is, however, a type of subluxation which is not apparently the result of traumatism but of systemic infection, such as rheumatic or scarlet fever, and rather more especially of upper respiratory tract infections. It is likely that this type of subluxation in a mild degree occurs more frequently than is believed, following such infections in children; and it is probable that in the past, before the roentgen ray diagnosis was possible or its technic fully worked out, many cases of limited motion or "wry neck" following cervical region abscesses and infections were in reality cases of atlanto-axial subluxation.

In a recent review Hess, Bronstein and Abelson[1] collected from the literature twenty-two cases of atlanto-axial subluxation of nontraumatic origin to which they added two personal cases. There are a few more such cases not included in these authors' bibliography.

In reporting a personal case of my own of this kind, resulting evidently from a retropharyngeal abscess, I do so, not because I can add anything of importance to what is already known and has been written on this subject, but with the desire of calling it to the special attention of otolaryngologists as something that especially concerns them. So far as I have been able to find, all of the important literature on nontraumatic atlanto-axial subluxation is to be found in general medical or pediatric journals. I have seen no report in any otolaryngologic journal. In my remarks, therefore, I shall touch especially on those aspects of this clinical entity which will be of particu-

*Presented before the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society, at Milwaukee, January 11, 1936.

lar interest to otolaryngologists, as it very frequently results from conditions that fall within their specialty.

So far as it known, anterior subluxation of the atlas on the axis, apart from a congenital deformity, can result only from rupture or relaxation of the transverse ligament. There can be a unilateral anterior or posterior displacement of the atlas due to a ruptured or relaxed transverse spinal ligament as well as an independent lateral or bilateral rotation, although usually the subluxation and rotation occur together. A ruptured transverse ligament is always the result of traumatism, but apparently its relaxation, permitting slipping and dislocation of the atlanto-axial articulations, may follow various conditions. It should be observed that subluxation of the joint is always associated with rupture or relaxation of the transverse ligament. There may be a rotatory deformity of the joint without relaxation of the ligament. In nontraumatic subluxation of the atlas the vast majority are of the forward or anterior type and are accompanied by some degree of lateral rotation. Berkheiser and Seidler[2] express the opinion that anterior subluxation predominate because the center of gravity of the head is located farther forward than the anatomic support.

The interesting thing in these nontraumatic cases, as pointed out by Jones,[3] is that the one predisposing factor which is constant is the existence of an inflammatory focus in the upper neck. Some French authors have expressed the opinion that the original infection was necessarily nasopharyngeal and, therefore, coined the term "nasopharyngeal torticollis" for the atlanto-axial subluxation; but it was found later that any infection which would cause hyperemia in the region of the base of the skull could give rise to the condition. Tonsillitis, pharyngitis, middle ear disease, mastoid disease, inflamed cervical glands and so on, would fulfill this condition. The nature of the infection, whether pyogenic, syphilitic or tuberculous, would be of no significance.

The mechanism by which diseases like those named cause relaxation of the transverse ligament is not, however, a matter on which there may be complete agreement.

The literature on cervical region abscesses and abscesses in the retropharyngeal space shows how closely interwoven are the lymphatic connections of those regions and how readily an infection may spread, from the middle ear or retropharyngeal space to the bones of the atlanto-axial joint. It is important to note that the atlanto-axial joint drains into the retropharyngeal lymphatic glands into which the

nasopharynx also drains. But other factors besides spread of infection seemingly are necessary before subluxation can take place. These are especially hyperemia in the region and decalcification of the bone. It is generally admitted that if there is a focus of infection anywhere in the upper cervical area, the field of hyperemia due to it will generally include the upper cervical vertebræ and, as Jones points out, particularly that part of the atlas which is nearest to the focus of infection. In pharyngeal infections the nearest point of the atlas is the central part of the anterior arch, and in mastoid infections the part of the atlas nearest to the infection is the lateral part of the arch on the same side as the mastoid.

In 1931, Greig,[4] of Edinburgh, published some personal investigations which proved that hyperemia causes decalcification of a bone and with the establishment of such a fact one can follow how spontaneous subluxation of the atlas and axis might be effected. After a short period of infection, say, from seven to ten days in the case of a pharyngeal infection, the amount of decalcification of the anterior and lateral part of the ring of the atlas is such that the arch of the atlas becomes softened and no longer affords a secure attachment for the transverse ligament, which is mainly responsible for maintaining the odontoid process in close relation with the atlas. Under such conditions even a slight movement may avulse the ligament and permit an anterior displacement of the atlas. With slight avulsion the subluxation is incomplete and gives rise to the clinical syndrome of atlanto-axial subluxation commonly observed; with complete avulsion there may be medullary interference and probable death of the patient. Cases of sudden death from atlanto-axial subluxation have been recorded. In any case the condition is always most serious, as the cord lies in a very restricted space between the odontoid and the back of the atlas and foramen magnum. A very slight exaggeration of the existing condition of subluxation may cause death.

Decalcification and relaxation of the transverse ligament as causative factors are favored by the clinical observation that atlanto-axial subluxation occurs mostly in children under twelve years of age in whom the bones have not yet reached maturity and are very highly vascularized. It may, however, occur in adults, as in Swanberg's[5] case. There are other interesting etiologic theories, however, such as that of Wittek,[6] supported by Jacobs[7] and others, who considered that the subluxation was the result of overdistension of the ligaments brought about by inflammatory metastatic effusion into the atlanto-axial joint. This suggests an inflammation of the bursa between the

odontoid process and the transverse ligament so that the letter softens and stretches. It is the theory of distension subluxation.

Similar to spontaneous subluxation of the hip, sometimes seen complicating the exanthemata, typhoid and mastoiditis, this condition may be due to joint effusion, capsule and ligament distension and pull in adduction. In the neck we have distension of the capsule of the atlanto-axial joints plus anterior disbalance of the head plus anterior neck muscle pull.

Grisel[8] emphasizes as a causative factor spasm and contraction of the prevertebral muscles associated with lymphatic infection of the retropharyngeal space secondary to some inflammatory condition of the nasopharynx.

In the great majority of the reported cases of nontraumatic atlanto-axial subluxation, the patients had inflammatory processes about the head and neck shortly before the onset of the subluxation, such as an acute tonsillitis, mastoiditis, pharyngitis, ulceration of pharynx, cervical abscess, acute dental infection, coryza, influenza, etc. Many of the earlier reported cases followed acute rheumatic fever with throat involvement. The history usually given is that after about a week or ten days' illness the patient suddenly developed a "crick in the neck" and kept the head twisted avoiding movement. The head is held rigidly, slightly in front of its normal plane, the occiput rotated toward one shoulder with the chin close to the neck and pointing toward the opposite shoulder. It is, as a matter of fact, the position of an ordinary wry neck except that there is no contracture or spasm of the sternomastoid muscle, the rigidity being evidently due to spasm of the deep cervical muscles. The tilting of the head is toward and the rotation away from the side on which the atlas has moved forward, and the spine of the second cervical vertebra can be palpated on the opposite side.

The three main clinical features are, therefore, rigidity, frequency of deformity, fixation and pain on movement. Swanberg points out also that every anterior dislocated atlas that he had observed showed one clinical sign, namely, that the posterior nasal pharynx was greatly reduced in size because of the projecting anterior arch of the atlas and this can be palpated.

An acquired torticollis with muscular spasm, following in the wake of an infectious disease, particularly of the upper respiratory tract, should at once arouse suspicion of subluxation or at least of a rotatory deformity of the atlanto-axial joint. Otolaryngologists

especially should be on the lookout for this complication. The clinical diagnosis can be confirmed by roentgenograms. The roentgenologic examination, if subluxation is present, should show in the lateral view an increased anterior atlanto-dental interval. There is a marked lessening of the shadow of the anterior part of the atlantoid ring compared with the bones lower down, shown in the anteroposterior roentgenogram.

My personal case is peculiar in that it followed a retropharyngeal abscess, the peculiarity merely consisting of the fact that I have not found a case with similar etiology among those reported in the literature, although one would imagine, *ceteris paribus*, that such an abscess would be a frequent cause of atlanto-axial subluxation, seeing the facility with which infection in the retropharyngeal space can be communicated to the atlas. In a case reported by Piotet,[9] in a girl of eight years, a torticollis following adenoidectomy, not dependent upon subluxation of the atlas, was relieved by treatment of a hitherto undisclosed complicating a small retropharyngeal abscess. The history of my case is as follows:

REPORT OF A CASE.

CASE 1.—F. G., male, white, age 9 years, entered Michael Reese Hospital on June 26, 1935.

Present History: The mother states that the boy was apparently well until eleven days ago when she noticed that he seemed less lively than usual and somewhat pale. This prompted her to take his temperature which registered 100° F. The next day, being apprehensive of an acute infectious disease, she examined him carefully and noticed the cervical glands on both sides slightly swollen and tender. He denied any sore throat, difficulty in swallowing or breathing. The temperature in the interim varied between 101° and 103° F.

Previous and Family History is irrelevant.

Examination on admission reveals an acutely ill child, lying on his back with his head inclined toward the left shoulder. The cervical glands on both sides are enlarged, firm, discrete and tender. There is slight rigidity of the neck.

Examination of the throat shows a slight bulging on the left side of the pharynx. A moderate amount of trismus is present.

Blood examination shows R. B. C. 4,530,000, W. B. C. 15,650, polymorphonuclears 81 per cent, eosinophiles 1 per cent, basophiles 1 per cent, lymphocytes 13 per cent, mononuclears 4 per cent.

Urine analysis, normal.

June 27, 1935. The only apparent change in the previous findings is that the bulging in the left side of the pharynx is larger and fluctuation is evident. The abscess was opened and considerable pus evacuated. The usual postoperative treatment was instituted and the child's progress to normal was uneventful with the exception of a marked left-sided torticollis. He was discharged from the hospital July 2, 1935.

An x-ray of the cervical region made through the open mouth shows an eccentric position of the odontoid process with reference to its ring and shows an actual tilting of the base of the skull and atlas with reference to the axis. There is no sign of breakdown at any point in the cervical spine.

Diagnosis: Left-sided retropharyngeal abscess causing subluxation of the atlas on the axis.

Treatment consisted of recumbency in hyperextension and head traction with a Sayre sling. The head of the bed was elevated eight inches. Hyperextension of the neck was maintained by a small neck roll and no pillow was used. When the spasm and pain ceased and the roentgenogram showed restoration of the anatomic atlanto-axial relationship a plaster cast was applied from the hips to and including the chin and occiput. The child made a good recovery and at the end of six weeks there was no clinic or roentgenologic evidence of the torticollis.

In treatment Berkheiser and Seidler recommend placing the patient on a Bradford frame, which immobilizes the whole spine, and applying a slight, constant traction to the head so as to obtain hyperextension of the head and neck. The time for reduction may vary from eight days to five months. Not only do the movements of the vertebræ when hyperextended represent the reverse of the movements by which the subluxation was effected, but recurrence of the deformity is prevented by the tension produced on the anterior spinal ligament which then acts as a splint to the vertebræ. Cessation of the infectious process and the concomitant hyperemia will check decalcification, which in the case of children should rapidly correct itself.

SUMMARY.

Nontraumatic atlanto-axial subluxation may follow a systemic infection but more particularly an inflammatory process in or near the upper respiratory tract which involves the cervical region in children.

Only a comparatively small number of cases have been reported in the literature, but the condition is probably more common than is believed, and, in a mild form, is often considered as wry neck.

The most likely etiologic factor is a relaxation of the spinal transverse ligament which is dependent on decalcification of the atlas by some infectious process.

A case originating from a retropharyngeal abscess is reported.

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XXI.

A SATISFACTORY METHOD FOR THE IRRADIATION OF MALIGNANT DISEASE OF THE ESOPHAGUS.

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The diagnosis of malignant esophageal disease is made in the majority of cases only when the neoplasm has become well established and after many discouraging results with various types of treatment it has been more or less generally concluded that conservative therapeutic measures seem most effective. In the past, intraesophageal radium has been used quite extensively, but difficulty has been encountered because of the inability to maintain the position of the radium at the site where irradiation should have its fullest effect.

A method which has proved satisfactory in maintaining the position of radium when properly placed and has allowed an accurate distribution of radiant energy is herein described.

A gastrostomy is performed as soon as the diagnosis of malignant disease of the esophagus is established. When the patient's condition is satisfactory following this operation, retrograde bouginage and graduated dilatation is performed until the lumen of the esophagus at the site of the neoplasm is large enough to admit the applicator which carries the radium. The radium applicator, as illustrated, is placed in position by the same method employed in passing retrograde dilating bougies. It is pulled into position by traction from above, and after determining by tactile sense and fluoroscopy the area to be irradiated the applicator is fixed into place by inflating the rubber attachments at each end with a 20 per cent sodium iodide solution. The iodide is introduced through a small rubber tubing which is attached to the distal end of the applicator and communicates through its shaft with the rubber balloon attachments at each end. When the proximal and distal rubber globes are inflated, the applicator assumes, roughly, the shape of a dumbbell. With the irradiating segment held snugly within the neoplasm, both extremities of the applicator are

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1. Roentgenogram showing a radium applicator with bulbous ends fixed in position at the site of the lesion.

Fig. 2. Roentgenogram taken 24 hours later showing a change in the position of the applicator, despite the fact that it was firmly fixed above and below. Arrow marks site of lesion.

Fig. 3. Roentgenogram demonstrating the radium aplpicator fixed into the site of the lesion by using the inflation method at its upper pole. Arrow marks the site of lesion. Roentgenograms taken 24 hours later show the applicator held in its original position.

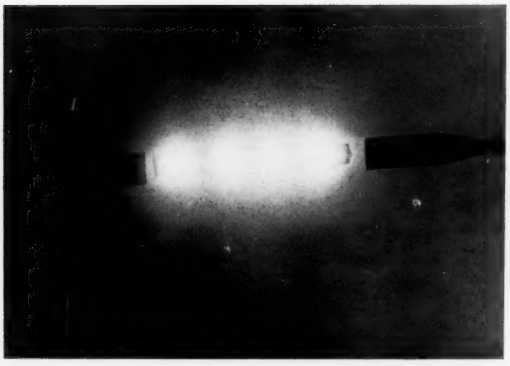

Fig. 4. Roentgenogram of the bougie revealing the amount of surface which is active during the process of irradiation. Four, twenty-five milligram capsules are outlined by their own irradiation. Radium needles or cells may be used to advantage.

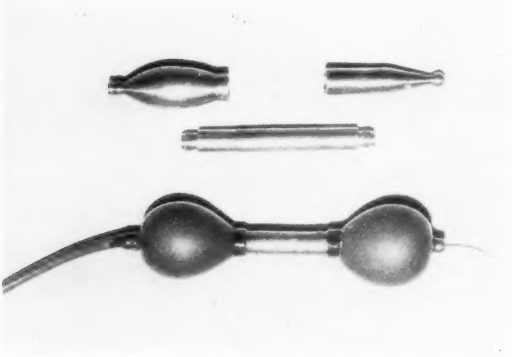

Fig. 5. Photograph showing component parts of the radium applicator assembled with rubber parts at each end inflated as it appears when seated in the esophagus.

inflated into normal esophageal lumen, thus holding the radium shaft firmly in position. The rubber tubing through which the sodium iodide is introduced and which also carries in its lumen the cord attached to the distal end of the applicator, is clamped and loosely fixed to the skin over the epigastrium. The cord attached to the proximal end of the applicator is fastened to the side of the face or about one ear with a strip of adhesive. The employment of this technic and this apparatus assures more or less constant irradiation in a fixed position within the lumen of the esophagus. Frequent postoperative fluoroscopic examinations give proof of this fact.

The radium applicator, which is five inches long, is constructed entirely of brass. The diameter of the barrel carrying the radium is three-eighths of an inch, which is increased about one millimeter by the addition of a layer of cellulose nitrate used to filter out the secondary irradiation from the metal. The length of the barrel is three inches, and therefore affords that length of irradiating surface. The proximal piece of the applicator is tapered and slightly blunted on the end to insure safe passage through the esophagus. It also gives attachment to the cord by which traction is exerted on it. The distal endpiece is ovoid in shape, its greatest diameter bing eleven-sixteenths of an inch. This bulbous end prevents the applicator from passing through that portion of the esophagus occupied by neoplasm and thus informs the operator when the applicator is properly seated. Both end pieces screw on to the barrel and likewise carry the rubber pieces which, when inflated with a small degree of pressure, each have a diameter of about one and one-half inches.

Radium in any form that gives a uniform distribution of irradiating surface can be used. The use of radium cells or needles, we believe, offers some advantages because they lie very near the center of the applicator and allow for greater filtration and increased distance from the radium source to the surface of the lesion. The massive single dose system or the divided dose method of irradiation may be used. This applicator lends itself admirably to the latter form of treatment because of the ease with which it can be inserted into position. We believe that large doses of irradiation are required in these cases, and in this connection it is our practice to administer a total dosage of 1,200 milligram hours per inch of irradiating surface, given as a maximum dose over a three or four day period.

60 WYOMING STREET.

XXII.

TELANGIECTASIA OF THE NOSE: TREATMENT BY MICRO-INJECTION OF SCLEROSIVE FLUID.

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The nose and flush areas of the face are favorite sites for the development of telangiectases. These acquired vascular dilatations arise by the enlargement of pre-existing vascular channels. Many stimuli act to cause chronic stasis of these surface vessels. A partial list includes skin diseases, alcoholism, toxic disorders, hypertension, exposure to the elements, and endocrine disturbances.

The most frequent cause is acne rosacea, a skin disease of varied etiology.[1] With the habitual use of alcohol, many men develop a severe type of acne accompanied by dilated capillaries. However, in other instances, telangiectatic lesions appear on the nose and flush areas of the face without any preceding lesions. Oftentimes these men are confused by the laity and even by the profession with chronic alcoholics. The embarrassing legend which has grown around red noses has caused many sufferers to seek relief in the past. However, no satisfactory method has as yet been devised which effectively removes this facial blemish.

The abnormal channels may be at the sides of the nose or at the base. In the latter location they are found at the apex or over the *alæ nasi*. Often the vessels cross the midline to interweave with those of the opposite side. Sometimes an amazing profusion of dilated capillaries gives the entire organ a reddish hue. In all cases the basic lesion is the enlarged blood capillary and its removal will effect a cure.

In 1934 I described a micro-injection technic for the eradication of telangiectases associated with varicose veins.[2] The success of this treatment on legs led me to increase its range of usefulness. Accordingly dilated capillaries were injected on the chest and neck, and eventually on the face and nose.

The technical barriers to the application of this treatment on the nose were twofold. In the first place, a needle had to be developed which was fine enough to enter the capillary lumen and yet possess enough rigidity to penetrate the thick epidermis of the nasal

Fig. 1.

skin without bending. Secondly, a fluid had to be found which would sclerose the capillaries without arousing unsightly pigmentary reactions. After the technical barriers were overcome, a physiologic problem presented itself because of the peculiar arrangement of the blood supply to the skin of the nose.

The venous ramifications of the blood vessels in the skin of the nose are exceedingly complex. The bloodstream may flow into the external jugular by way of the anterior facial vein or communicate with the ophthalmic vein or pterygoid plexus. Through these channels an amazing network of other communications is possible, but eventually the course of the blood stream is into the internal jugular vein. For theoretical considerations it is preferable to divert the course of the injected solution away from the angular vein of the nose toward the anterior facial vein and thus down to the external jugular vessel. This is accomplished during a treatment by pressure on the root of the nose over the angular vein. Simultaneously the patient is told to inspire deeply, thereby creating an increased suction in the thoracic cavity. This directs the bloodstream down by the relatively shorter external jugular route.

Practically it is my experience that the treatment may be given without any of the above precautions. The few minims of hypertonic saline solution that are injected are instantly diluted by the blood and rendered inactive to neighboring vessels. Clinically the localization of sclerotic effect can be noted by the fact that the reaction is always limited to one or two capillary loops. Widespread reactions never occur and no neighboring tissue is ever affected.

Fig. 2.

The result of the injection upon the dilated capillary is in one of two directions. Usually a gradual shrinking and disintegration occurs. Occasionally a definite thrombotic streak is observed after a few days which is ultimately absorbed. The first type of reaction is one that is peculiar to the nose. I have not observed it on legs. Sometimes an immediate vasospasm takes place which persists until the capillary disintegrates. The final result in any event is the clean removal of the offending blood vessels.

TECHNIC.

The procedure can only be done under strong illumination and magnification. To this end a powerful floor lamp and a good binocular loupe must be used. The solution of greatest utility is 30 per cent sodium chloride in sterile distilled water. The average amount of fluid injected varies from two to eight minims. This eradicates any type of nose capillary.

The needle is always started in healthy tissue and the point carried toward the dilated vessel, keeping directly below the epidermis at all times. The capillary loops are firmly fixed in the relatively dense and adherent skin of this region. With a gentle push it is singularly easy to enter the lumen of the vessel. This entry can be felt as a lack of resistance as the needle point pierces the vessel wall. As a further proof of entry, the needle point can be wriggled from side to side or moved farther into the canal of the vascular channel. Blood can usually be aspirated from the larger vessels. This aspiration is sometimes difficult in the smaller channels, although a show of blood may be obtained even here by prolonged suction. As the

fluid is injected the capillary loops become blanched and change in size. The course of the injected fluid can be traced by this phenomenon and the amount of sclerosis thus controlled.

No cramping is felt with this technic, although a momentary burning or stinging sensation may be felt by the patient. This, however, soon passes off with no succeeding discomfort. The prick of the needle entry is of little consequence with this fine technic, and altogether the method is surprisingly free from discomfort.

The patient is treated in the sitting position. While the injection is being made, compression may be applied with the finger tips over the root of the nose. At the same time the patient may be asked to raise his arms above his head and inspire deeply. Following injection, a little pressure will control oozing from the needle hole. No dressing is used. Treatments are given twice weekly and continued until all segments are obliterated.

The illustrations show the progress of a case receiving this treatment. The second photograph shows the lesions completely removed. The nose of the patient illustrated was a definitely red one at the beginning of the treatment. However, at the termination, when the network of dilated capillaries was completely removed, a normal nose resulted.

CONCLUSIONS.

1. Telangiectasia of the nose is an embarrassing lesion that has had no satisfactory therapy.
2. A micro-injection technic is described which is based upon the treatment of similar lesions on the legs.
3. A special needle is employed to inject 30 per cent sodium chloride directly into the capillary lumen.
4. The method is safe and cosmetically good.

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XXIII.

THE TREATMENT, OTHER THAN OPERATIVE, OF THE NASAL ACCESSORY SINUSES.*

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CINCINNATI.

The treatment of chronic nasal sinus disease in a nonoperative manner is, in many instances, an unsatisfactory procedure. We must, therefore, when attempting this method of treatment, select our cases with discretion and not continue to treat for any length of time a patient who is in need of a more radical procedure. We shall eliminate immediately from our discussion the treatment of all very acute inflammatory processes of the nose and nasal accessory sinuses, associated with or without fever. We believe that any local treatment at this time, unless it be the application of a vasoconstricting agent, will only kindle the fire and be the means thereby of causing a reaction with a spread of the disease.

The chief consideration will, therefore, be the subacute and chronic infections. How shall these patients be treated nonsurgically? The various means and methods of treatment which we have employed in our practice are the following:

Systemic,
Treatment of the nasal mucosa,
Median rhinoscopy,
Treatment of nasal recesses,
Tampons,
Suction with irrigation,
Dilatation of the ostia and nasofrontal duct,
Irrigation of the sinuses,
Infra-red and ultra-violet therapy.

In approaching the subject from the systemic point of view, let us first have something to say regarding the endocrine glands. We are all in agreement with the thought that a long-continued toxemia originating in the nasal sinuses, or elsewhere, has an exhausting effect

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on the thyroid gland, as well as other ductless glands. The thyroid gland must be looked upon as the great activator of metabolism, so that by supporting this gland with a tolerant dose of thyroid extract, other opponent glands, such as the suprarenal and pancreas, also become more atcive, and in consequence there takes place a proper balance by indirect means. In giving thyroid extract, we should always remember an important axiom, namely, the more the patient requires the drug, the smaller should be the initial dose. The reason for this is quite obvious if we but remember that infiltration of tissues with mucin has taken place as the result of under-action, and that rapid liberation of mucin by a large dose of thyroid extract will bring too much pressure to bear on the excretory organs and intolerance results.

A patient with benign hypothyroidism usually has a subnormal temperature between 4 and 6 p. m. In the administration of thyroid extract the afternoon temperature should be our chief guide. In our estimation, it is of greater clinical importance to consider the presence of hypothermia than it is to rely on the basal metabolic rate in the administration of thyroid extract, in view of the fact that there are many patients with a benign hypothyroidism who do not have a minus rate. When the basal metabolic rate is minus, there are usually other symptoms of hypothyroidism present which make us reasonably certain of our diagnosis. By beginning with 1/10 of a grain of the extract three times a day and increasing the dose every three or four days until the afternoon temperature becomes normal, one can readily ascertain the proper tolerant dose for each patient. Most patients require 2 to 3 grains of thyroid extract daily; a few have taken as much as 6 to 8 grains a day. When the tolerated dose has been reached, it should be given three weeks out of every month for a long period of time. One of the first signs of intolerance is the presence of coryza, probably caused by the iodine content of the thyroid extract. When this occurs the thyroid medication should be stopped for three days. We should also assume that the tolerant dose has been reached when there is a loss of body weight, and when the pulse rate, which has been slow, reaches 95 beats in a minute.

Some patients with benign hypothyroidism are thin and have a rapid pulse.[1] These individuals will tolerate small doses of thyroid extract if the drug is given cautiously. Response to treatment is very often our only guide to a correct diagnosis of the hypothyroid state. This is exemplified in women who suffer with headaches during the menstrual period, and in whom the symptoms disappear immediately after menstruation ceases. There seems to be an aggravation of the

hypothyroidism at this time, which is of such a benign character that it causes few, if any, symptoms during the remaining days of the month.

There is another systemic derangement which is many times associated with hypothyroidism. I refer to chronic intestinal toxemia, the treatment of which requires our attention if we wish to obtain a beneficial result from the thyroid medication. Clinical experience has taught us that many of the congestive states of the nasal and pharyngeal mucosa are caused by toxic material originating in the colon. The average individual does not regard his diet seriously. He eats more than his system requires. As a result, a toxic state of the blood is present and the mucous membrane of the nose is congested. When these patients are given free catharsis or colonic irrigations, and are placed on a diet free from meat and containing a minimum amount of salt, the nasal mucosa may assume a normal color and hypersecretion of mucus may become much lessened.

Intestinal stasis, without doubt, has a deleterious effect on the thyroid gland in the form of under-action, and when this takes place the mucous membrane of the nose may assume a pale color. Daily observation has fully convinced us that color changes of the mucosa are present as frequently in the nasopharynx as they are on the septum. We have seen patients with pale septa in whom the nasopharynx, especially in the region of the eustachian tube ostia, presented a white infiltrate. These patients were hypothyroid individuals, and their nasal mucous membrane returned to normal color after they were given the tolerant dose of thyroid extract and treatment was directed to the intestinal toxemia. We do not hesitate to assert that the hypothyroid state and chronic intestinal toxemia are, without doubt, the most frequent systemic derangements that require the attention of the rhinologist. There are other diseases, however, such as anemia, syphilis, diabetes and the gouty-arthritic diathesis that may cause changes in the sensitive nasal sinus mucous membrane.

To sum up this part of the subject, we may rightly say that the most difficult problem before the medical profession today is the study of the constitution of the patient, inasmuch as each individual reacts to certain irritants according to the character of his inner qualities, and these qualities we term constitution. It is our belief that the treatment of the constitution of the patient with diet, hygienic measures of living, endocrine and intestinal therapy and the examination of the blood for various changes is of greater importance

than the detection of the type and virulency of the bacteria. To emphasize the bacterial aspect only and give vaccines, specific or non-specific, is not sufficient. We must not forget that the bacteria would probably not have been active if the constitution of the patient had not been ready to receive them. A patient in whom the constitutional elements are inadequate to combat infection readily acquires a chronic state of the nasal sinus mucosa following an acute infection. In these patients two physiologic functions are easily lost, namely, movement of the ciliated epithelium and secretion of mucus, both very important functions when infection takes place.[2] A patient whose tissues are unable to perform their physiologic function at the time of an infection is left with an edematous nasal mucous membrane which may cause retention in the recesses—a favorable condition for the establishment of chronocity.

The mucous membrane of the nasal sinuses may be characterized as weak, pale, thin, easily detachable and poorly nourished; just the opposite to the predominating physical traits of the parent cavity. The frailty of the one and the hardihood of the other is to be sought in the correlation of growth and function. An infection evanescent in the nose may very easily overpower the mucous membrane of the sinus. It behooves us, therefore, to insure free nasal respiration, and this, as we all know, very often requires nasal surgery for its accomplishment.

Much relief can be given by doing a septum operation. It is our contention, however, that too many septum operations, and too many middle turbinectomies are performed without giving the nasal sinuses due consideration at the time of the nasal operation. If this fact is overlooked, a severe reaction may follow the septum operation and an acute flare-up of a chronic nasal sinusitis may occur.

To give the patient better breathing space, it very often becomes necessary to infract the inferior turbinate towards the lateral wall of the nose, to remove posterior hypertrophies or to excise, if necessary, the overhanging edge of the inferior turbinate. Vasomotor rhinitis may often be controlled by ionization of the nasal mucosa, according to the method of Warwick, and in a milder way by the application of trichloracetic acid to the four points of Killian—namely, the tubercle of the septum, the lateral wall of the nose opposite the middle turbinate, the anterior portion of the septum, and the anterior third of the inferior turbinate. We have also found the application of diathermia, at times, to be of value.

There is no more satisfactory procedure to be used in the conservative treatment of a patient suffering with headache, as the result

of a mild, chronic ethmoiditis of the serous type, than median rhinoscopy. To make a median rhinoscopic examination, the middle turbinate is infracted towards the septum with a long-bladed Killian speculum. When this is done, a good view of the ethmoid floor may be had, so that pathologic changes as well as anatomic variations may readily be seen. Following the infraction, the floor of the ethmoid, as well as the lateral wall of the nose opposite the middle turbinate, is cauterized with trichloracetic acid. We wish to recommend this simple procedure, especially in cases of subacute ethmoiditis in which the headache is not the result of the nasal sinus inflammation per se, but is caused by pressure of the middle turbinate against the lateral wall of the nose, probably irritating in this way the lateral branches of the nasal nerve.

Following an acute infection in the nose there may occur a localized inflammation in the recesses—namely, the hiatus semilunaris, or in the recessus spheno-ethmoidalis. When the infection has localized itself in the former it simulates maxillary sinusitis, especially if the hiatus terminates in a concavity on the lateral wall of the nose. Hiatus suppuration is very difficult to differentiate from antrum suppuration, but may be suspected when, upon irrigating the antrum, the fluid returns clear except for a small amount of pus at the end of the washing. Local applications of a silver preparation or iodine solution should be made to the region of the hiatus and lateral wall following the irrigation. The recessus spheno-ethmoidalis is situated above the superior meatus and forms the interval between the posterior extremity of the supraturbinal and the anterior aspect of the body of the sphenoid. It is a shallow but well-defined triangular fossa of much clinical significance, and is to the posterior group what the hiatus semilunaris is to the anterior group. A patient with an infection of the mucous membrane covering the recessus spheno-ethmoidalis usually gives a history of having had a severe cold in the head which did not entirely disappear. The nose feels stuffy, the sense of smell is affected, the olfactory fissure is edematous, and very often a plug of mucus may be seen hanging from the posterior end of the middle turbinate, upon examination with the nasopharyngoscope. In order to treat the recessus spheno-ethmoidalis, repeated applications of a 20 per cent solution of cocain must be made to the edematous olfactory fissure, after which a cotton-wrapped probe, bent at right angle and saturated with a 20 per cent cocain solution, is applied to the region of the posterior end of the middle turbinate, and then passed upward into the recess. After cocainization, applications are made to the recess with Lugol's solution, followed by a nebula of Colet's solution.

We do not have much faith in the use of nasal tampons saturated with silver preparations. These tampons act by osmosis and are just as efficacious when moistened with hypertonic salt solution. Tampon treatment with a colloidal silver preparation, if employed, should be used only in the first few weeks of the infection. It is, in our opinion, inadvisable to use this form of treatment for a longer period, inasmuch as we have seen four patients with argyria in the past few years who stated that a silver preparation had been used in the nose for a period of time varying from six months to a year and a half.

Suction with irrigation is of great benefit in subacute cases, especially in children.

We come now to the consideration of the treatment of the nasal sinuses themselves. The methods employed are dilatation of the ostia, irrigation of the cavity and light therapy. Patients who suffer with so-called "vacuum headaches" may be relieved by gradually dilating the nasofrontal duct with Ritter's sounds. These patients may have an anatomic variation of some kind, such as apposition of the bulla ethmoidalis to the uncinate process, tortuous nasofrontal duct, or upward displaced infundibular cell, which, when broken through by the sound, allows for the passage of air and relieves the headache. Pathologic changes may or may not be present in these cases. Dilatation of the normal ostium of the maxillary sinus has given relief to patients complaining of a fullness and pressure in the antrum region and in whom there was apparently no pathologic change present within the cavity. The same may be said regarding the sphenoid cavity. The ostium of this sinus is often closed because of edematous thickening of the anterior wall. Dilatation of the opening, followed by the application of trichloracetic acid to the anterior wall, will often bring relief of what appears to be a vidian neuralgia. To treat the anterior wall of the sphenoid, it is often found necessary to infract the middle turbinate towards the lateral wall of the nose.

Irrigation of the frontal sinus is not always possible without widening surgically the nasofrontal duct. Irrigation should never be carried out in the presence of a very acute inflammation, for fear of causing a severe reaction which may subsequently develop into an osteomyelitis. We prefer, as an irrigating fluid, the use of a 1.0 to 1.5 per cent solution of calcium chloride because of the beneficial action of this drug on edematous tissue. When the sphenoid cavity is to be irrigated, it is advisable before inserting the cannula, to sound the cavity with a probe slightly bent at its end. The ostium is easily

reached in most cases if we follow Zuckerkandl's line, which is the extension of an imaginary line drawn from the anterior nasal spine to the center of the lower edge of the middle turbinate. When vidian neuralgia is an accompaniment of sphenoid sinus disease, the instillation of a 10 per cent novocain or cocain solution, after irrigation, often relieves the pain.

An acutely inflamed maxillary sinus should rarely, if ever, be irrigated in the presence of fever, because of the danger of causing a systemic infection following the lavage.[3] Patients are usually able to care for the infection in an antrum, provided no other irritant appears, such as the irritation which may be caused by an antrum lavage. As soon as another insult is added to the infection already present in the antrum, an imbalance of the body mechanism takes place. The bacteria gain the upper hand, and many times, because of the injury to the mucous membrane from puncture, a secondary inflammation ensues and a local or general sepsis may be the result.

Patients presenting themselves at the office with the history of a recent acute attack of antrum infection are given an intramuscular injection of a nonspecific protein remedy for three successive days, in an attempt to bring up their body resistance before irrigating the cavity. We prefer omnadin for this purpose. When fever occurs after irrigation, or if there is present a fibrinous deposit in the region of puncture, further lavage is abstained from for at least two or three days. Proof puncture in the middle meatal wall is, in our minds, more simple of performance than the one made under the inferior turbinate, and is our method of choice. When the cavity is entered beneath the inferior turbinate, one must employ a sharp cannula, which may readily injure the plexus of veins which are usually well marked on the lateral wall of the nose along the course of the lacrimal canal. Furthermore, a sharp pointed trocar is more apt to injure the engorged veins present in the thickened mucosa lining the antrum cavity. Injury to the veins not only makes it possible for air embolism to follow puncture, but also predisposes to the development of a septic thrombophlebitis with subsequent pyemia. There is less likelihood of injury to the veins if the cavity of the antrum is entered in the middle meatal region with a blunt pointed cannula on account of the thinness of the wall in this region and the fact that very often puncture is found unnecessary because of the presence of normal or accessory ostia. Ringer's solution, as modified by Fischer, is probably one of the best solutions to be used for irrigation, because of its calcium content. The following is Fischer's modification :

Sodium chloride	263.7 gms.
Calcium chloride (dry)	21.0
Potassium chloride	10.6
H_2O q.s	1000 cc.

Twenty-five cc. of this stock solution to be placed in 500 cc. of sterile water for irrigating purposes.

If the patient complains of having had severe pain following the lavage, we instill, after future irrigations, a dram of 2 per cent novocain solution. When there is present a saprophytic infection, all suspicious teeth should be extracted and the cavity irrigated with a solution of permanganate of potash.

Many times, in the treatment of an antrum infection, we are dealing with a mixed form of disease in which an infection has been supplanted upon an antrum mucosa which already has undergone pathologic changes. The acute infection disappears slowly, but an edematous mucous membrane remains. Such patients should be re-examined several weeks following the infection in order to exclude a chronic state of the mucous membrane which may require operative intervention.

Infra-red therapy is a valuable adjunct in many cases of subacute nasal sinusitis. Especially is this true in patients affected with subacute ethmoiditis. An exposure lasting from forty-five minutes to one hour is necessary if good results are to be obtained.[4] Supplementing this therapy we often use Bier's hyperemia. An elastic band about an inch wide is placed around the neck immediately above the clavicle and allowed to remain three hours. It must be applied sufficiently tight so that the patient experiences a sense of fullness in the head, and should be applied again one hour after its removal. The passive hyperemia thus established accounts for its beneficial action. Its use is contraindicated in the presence of vascular hypertension and in all patients in whom pus is retained under pressure. Ultra-violet radiation is also of benefit in some cases.

The treatment of nasal sinusitis in children is one of our most difficult problems.[5] Many factors must be given due consideration, such as diet, hydrotherapy, exercise, calcium deficiency, endocrine disturbance, intestinal toxemia, anemia, sugar shortage, allergy and infection originating in an adenoid. Before doing a tonsil and adenoid operation it is imperative to make a careful rhinoscopic examination and to take a skiagram of the nasal sinuses if there is a history of repeated colds with nasal discharge. Inasmuch as the maxillary sinus

is many times the chief offender, would it not be more to the point to direct our attention to this sinus at the time of the tonsil and adenoid operation? We have had very satisfactory results when this was done, and very often it required but one irrigation of the antrum at the time of the tonsil and adenoid operation to bring about a cessation of discharge.

The child must be taught to blow the nose in the proper manner and, if possible, the nasal drill, as advocated by G. Hickling,[6] should be tried. The drill is as follows:

1. Hold a piece of gauze beneath the nose.

2. Grasp the top of the nose between the eyes with the right thumb and forefinger, the elbow being raised to shoulder level, leaving the nostrils open.

3. Blow the nose, and at the same time bring the head forward and down, and depress the right elbow to the waistline, the top of the nose still being held.

4. Inspire and raise the head to the original position, repeating the process ten to fifteen times in a rhythmic manner.

In many instances, no matter what is done, the child continues having successive attacks of sinusitis and is slowly laying the seed for chronic sinus disease of adult life, at which time the sinus may be approached in a more radical manner. One way of preventing chronicity is to treat carefully the acute attack.

In conclusion, may it again be said that the nonoperative treatment of nasal sinusitis presents many interesting angles. In applying the various forms of treatment, both local and general, we must forget, for the time being at least, that we are rhinologists, limited to a small specialized field. We must try to be real doctors, who are making a special study of a very sensitive part of the body—the nose and its adnexa—which areas are affected very readily by a systemic dyscrasia.

An early surgical approach is, in our opinion, a great mistake. Surgery, in most instances, should be undertaken only after a careful analysis has been made, or should follow the conservative treatment when this has been of no avail. When surgery is necessary, its success will depend on the ability of the surgeon to detect every affected sinus and his accuracy in removing all of the diseased mucous membrane.

19 GARFIELD PLACE.

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XXIV.

RHINOLITHS: REPORT OF THREE CASES WITH A REVIEW OF THE LITERATURE.*

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AND
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Rhinolith, nasal calculus, or nasal concretion is an uncommon affection occurring in but few instances in one's practice of many years. The rarity of this condition has been pointed out by many authors. A review of the available literature since 1916 reveals less than forty recorded cases, but we believe that there are innumerable instances which have not been reported. Formerly rhinoliths have been observed with greater frequency, but in recent years it would seem that its occurrence has diminished. This is no doubt due to the greater number of routine nasal examinations and nasal corrections, which eliminates the cause of stasis within the nasal cavity, thereby lessening the prevalence of this condition.

Prior to 1921, Thomson[1] quotes Key-Aberg,[2] who found over 300 cases that had been reported up to that time. Bailey[3] has been able to collect forty-nine cases from the literature in a fifteen-year period (1910-1925). From 1925 to 1933, Morwitz[4] collected fourteen cases from the literature. Since 1933 we have found four additional cases and add three of our own, making a total of seventy cases reported from 1910 to 1935. As far as we could find, the largest number of cases reported by a single author is that of de Graaf,[5] who reported seven cases. Joukovsky,[6] in a large clinic, observed but four cases in twenty-five years.

In order to obtain data, informative of this condition, we selected thirteen consecutive cases from the more recent literature in addition to our three. A detailed review of the sixteen cases is presented.

Rhinoliths are usually divided into a true and false type. In the true form, there is an endogenous nucleus usually consisting of inspissated mucus, blood or crusts; while in the false type an exogenous

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nucleus is present in the form of a foreign body. The majority of rhinoliths are of the true type. In sixteen of the more recent cases we found seven of the true type and six of the false, and in three instances no detailed description of the stone was available.

It is noteworthy to point out that one would expect to encounter many more cases of this affection since the accumulation of nasal secretions, pus or crusts, is present in most individuals at one time or another. Although in the true form some alteration in the character of the nasal secretion, causing favorable conditions for the deposition of inorganic salts is said to be an etiologic factor in some instances, we believe that the presence of some nasal disease or deformity which produces varying degrees of nasal obstruction, resulting in poor drainage of the nasal cavity favoring stasis of the secretions is the most important factor in the formation of the true type of rhinolith. The inorganic salts from the nasal and tear gland secretions adhere to the nucleus, and as it enlarges it has a tendency to fill the nasal space, impacting itself in the surrounding tissues.

The nucleus of the true type of stone usually consists of bacteria, blood, leucocytes, mucus or scabs. In the false type the nucleus is a foreign body. This form is not as common as the true type. Our three cases, however, were all of the false type. A large variety of foreign bodies have been found in the nucleus, such as nut shells, beads, seashell, coal dust, fruit stones, vegetable matter, cement, hair, screws, buttons, splinters, etc. At times it results from the presence of a sponge left in the nasal chamber following some operative procedure or other form of treatment. The following foreign bodies were encountered in our three cases: gauze sponge, shoe button and a piece of twine. Foreign bodies may also be introduced through the nose during vomiting or choking or may penetrate the tissues from without, such as in case of particles of stone or metals from an explosion or from a bullet wound.

Deposits of inorganic salts of calcium, phosphates, magnesium and sodium chloride, form the greater part of the stone, especially of the true type. These salts are derived from the nasal and lacrimal secretions. According to Polisar[7] the salts originate mostly from the tears and to a less extent from the nasal secretions. The stones are frequently covered with granulation tissue. On section it is observed that the salts have deposited around a nucleus of some material, such as inspissated mucus, clots of blood, sequestrum from turbinates or septum or some other foreign body. The consistency of the stone varies. It may either be hard or brittle, friable, porous or may be soft

and crumbly and break easily. The color of the concretion varies from coal black, dark brown or grayish, to lighter shades. According to Bailey, it may resemble the appearance of an oyster shell.

Nasal calculi are usually single and occur on one side only. There is no case of bilateral calculi recorded, as far as we could ascertain. Bailey likewise found no instance of bilateral rhinolith, but also remarks that there is no case recorded where multiple concretions occurred in one side of the nose. It seemed to us, however, that there is no reason why multiple concretions cannot occur in one side of the nose if conditions were favorable.

The size of the stones vary considerably, from a small millet seed or granule to one of large proportions. The largest stone reported is that of Botey,[8] weighing 110 gms., about the size of a hen's egg. Melay[9] reported a stone weighing 85 gms., and others report stones of varying weights.

Any part of the nasal chamber may be involved, but they are most commonly found in the lower part.

Rhinoliths vary in shape, but usually conform to the space in the nose which they occupy. They are generally spherical and irregular, some showing prolongations. The stones may be nodular and rough or smooth surfaced and occasionally lamellated.

From this series of sixteen cases we find the male sex predominates. There were twelve males and four females. Our three cases were males. Seeligman,[10] in a collection of 100 cases, found the condition more frequent in females. There were sixty-two females, twenty-nine males and in nine no sex was recorded, a ratio of slightly more than 2 to 1. Key-Aberg likewise found the condition more prevalent in females in over 300 collected cases.

Nearly all cases have been in adults. In the sixteen cases, the ages ranged from six to sixty-nine years. Polisar reported the case of a boy of fourteen with a true nasal concretion. In one of our cases a false rhinolith was found in a boy aged six, which is the youngest thus far reported. The condition is rarely found in children, although in our opinion many of these cases begin in early childhood, but are not recognized until later in life.

Occupation is said to be an etiologic factor in some instances, in the production of rhinoliths. Some of the nuclei have been found to contain substances such as coal dust, cement, hair and fiber.

Nasal concretions are usually of long standing. According to Polisar, a rhinolith has been known to have remained in position for a period of over thirty years. The true type of nasal calculus is nearly always of long duration, while the false type may be either of long or short duration.

Unilateral nasal obstruction is usually found in these cases. A foul nasal discharge is commonly present. Epistaxis which comes on more frequently at night, is often noted, but is rarely severe. Foul breath, headaches and difficulty in breathing are common symptoms. Complications are not unusual. There may be pressure atrophy of the adjacent structures and subsequent decomposition; deviation of the septum to the opposite side and also destruction of the septum in rare instances. More rarely, as mentioned by Knight,[11] perforation of the palate and facial paralysis have been noted. Gorman[12] reported a case which revealed numbness and swelling of the face with deafness on the affected side. The involvement of the sinuses on the affected side was commonly observed in most of the reported cases. Pansinusitis of the affected side was present in two of our cases.

Many of the reported cases give no detailed x-ray description of the condition. According to Lobell,[13] x-ray is of no value in the differential diagnosis, but further states that if the base is of metallic nature the x-ray would be of immense value. We feel, however, that the x-ray is of great value, not only as a diagnostic measure of the concretion itself, but it is an important diagnostic method of visualizing the calculus and in revealing other associated conditions and complications which are frequently present in this condition. The calculi are usually visible, as a rule, because of its large opaque content. On carefully taken films much information may be obtained of the position, size and shape of the calculus as well as its effect upon the surrounding structures and involvement of the sinuses.

In the differential diagnosis, syphilitic sequestration, calcified polyps, osteomata, necrotic bone and malignancy must be considered. As a rule, the diagnosis is made with ease.

REPORT OF CASES.

CASE 1.—W. N., colored male, aged 30, was seen in the clinic of the Baltimore Eye, Ear and Throat Hospital in January, 1935, complaining of severe agonizing headaches, foul breath, copious, foul nasal discharge, and obstruction to nasal breathing on the right side for about two years. About six years ago a sinus operation was performed elsewhere. This was undoubtedly, from his description, a Caldwell-Luc operation.

On examination in the clinic the patient was seen to have a marked nasal obstruction on the right side. The inferior turbinate on this side was so turgescent

as to impinge on the septum. The swelling of the turbinate and the great amount of muco-purulent exudate blocked any view of the interior of the nose. The secretion was thick and foul. The turbinates were not only intumescent, but hyperplastic. In an attempt to shrink the nasal tissues a grayish white, bony looking structure was observed posteriorly in the right nasal cavity. When palpated with a probe it was felt to have a hard bone-like consistency. An attempt to pull on it with a pair of bayonet forceps revealed an immovable mass. The structure was considered as a possible sloughing vomer at that time. The Wassermann reaction was negative.

X-ray examination of the nasal sinuses revealed a marked clouding of the right antrum, suggesting evidence of a chronic infection On careful observation, a calcified shadow was noted encircling another shadow of lessened density. The wall of the nasal side of the antrum showed marked erosion with evidence of bone destruction. The nasal chamber on this side was also clouded. There is some deviation of the septum to the left. These changes indicated the presence of a large foreign body extending from the interior of the right antrum into the nasal chamber.

At operation the hard brittle structure found in the nose was grasped with a bone biting forceps and removed piece-meal. Examination of the specimen revealed a thick shell of brittle calcareous material surrounding a large, discolored, foul-smelling, gauze sponge. The sponge had evidently remained in situ since the patient's last nasal operation, some six years previously.

CASE 2.—C. S. D., white male, aged 26, complained for one year of redness of the left eye. He had been under the constant care of an ophthalmologist during this period, and the condition showed signs of improvement at times, but as soon as treatment was relaxed the eye condition returned with greater severity. Photophobia and lacrimation were constantly present. Because of the unilateral character of the ocular condition an examination of the nose was made despite a negative rhinologic history. The left nasal chamber was slightly narrowed by an anterior septal deflection. On careful observation a small plug of thick foul-smelling pus was seen in the lower meatus. Following the removal of the pus there was still a white looking object visible between the lower turbinate and the septum. The object, on palpation with a sound, felt like a stone. It could be pushed backward for a short distance toward the nasopharynx, but this procedure caused considerable pain. An attempt was made to remove the object, but due to the anterior septal deflection, this could be accomplished only after much of the calcareous matter was broken off with the forceps. The stone weighed 20 grains, was irregular in shape, having a round black body covered by chalky material. After breaking away the calcareous matter, the black body proved to be an ordinary shoe button. Within ten days following the removal of the rhinolith, the eye symptoms entirely disappeared. The patient had no recollection of ever having introduced a shoe button into his nose. Hence one must deduce that it was introduced during a very early period of childhood and had remained in the nose for a period of from 18 to 20 years.

CASE 3.—White boy, aged 6, complained of bad breath and a purulent discharge from his nose since the age of two. His tonsils and adenoids were removed and he was taken to several rhinologists without any relief from this condition. Examination revealed a foreign body in the right side of the nose. Investigation of the stone proved it to be a small calcified foreign body with a piece of knitted twine forming its nucleus.

CONCLUSIONS.

1. Three cases of rhinolith are recorded, all of which are of the false variety. The first case was due to a sponge retained in the nose following an operative procedure. The second case showed a shoe button to be the obstructing factor. In the third case, the nucleus was a piece of twine.

2. The first case is of unusual interest. The rhinolith due to a sponge was found to lie obliquely with one end in the antrum and the other protruding into the nasal chamber. A deposit of calcium surrounding the sponge made it radiable and clearly visible on the roentgenogram

3. The third case, as far as we can ascertain, is the youngest patient on record with a rhinolith.

710 MEDICAL ARTS BLDG.

We are greatly indebted to Doctor Lee Cohen, who kindly permitted us to report two of his cases.

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XXV.

PLASMA CELL TUMORS OF THE NOSE AND NASOPHARYNX.*

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Plasma cell tumors of the nasopharynx are reported rather infrequently in American medical literature. Claiborn and Ferris,[1] in 1931, in the Archives of Surgery, reported twelve cases obtained from foreign literature. They abstracted those cases and added two of their own. These tumors have been variously termed myeloma, plasmocytoma, plasmosarcoma, and malignant plasmoma. Their degree of malignancy varies, some of the reported cases being highly malignant while others appear to have run a benign course.

Ewing,[2] in his textbook on Neoplastic Diseases, in speaking of extramedullary plasma cell tumors, said:

> "Considering their comparative frequency and rather peculiar clinical course, they have received inadequate attention. The growths occur chiefly in the nasopharynx, alveolar borders, tongue, lips, and cervical lymph nodes, but they have been observed in many other regions. The course is relatively slow, but they may recur after extirpation and become associated with chronic cachexia. They are commonly classed as lymphosarcoma, but being as a rule, benign processes with very indistinct neoplastic properties, it is highly important that they should be separated from the malignant lymphosarcoma. The structure presents a diffuse growth of more or less typical plasma cells."

Kemp G. Cooper,[35] in his recent article "Plasmocytoma of the Paranasal Sinuses," writes the following:

Attention has been drawn in recent years to a considerable number of reported cases of a comparatively rare form of myeloma which occurs in various parts of the upper respiratory tract, resembling histologically the multiple myelomas commonly found in the long bones but differing clinically in many respects.

The growths are designated as solitary plasmocytomas and pathologically may be classified as malignant lymphomas, of which the multiple myeloma and solitary plasmocytoma represent sugbroups. The great majority of these solitary plasmocytoma have occurred in the upper respiratory tract, principally in the nose and nasopharynx,

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and because of their solitary nature they have been particularly amenable to treatment. In a few of the reported cases, plasmocytomas occurring in this locality were associated with metastasis and followed the usual course of malignant tumors.

Cooper then reported two cases, one requiring a radical external approach for the complete removal of the growth.

In reviewing the literature, I have found that the article by Claiborn and Ferris[1] was most comprehensive on this subject. I shall therefore quote quite freely from their article (with the personal permission of Dr. Claiborn).

After abstracting from foreign literature twelve cases of plasma cell tumors and reporting two of their own case, Claiborn and Ferris make the following comment:

"While the series of cases that we have collected is too small to justify any general conclusions as to the age and sex incidence in cases of tumors described, it is interesting to note that of the fourteen cases, twelve occurred in males and only two in females. The youngest patient was twenty years of age, while the oldest was sixty-nine. Of the fourteen patients twelve were forty years or more of age. The predominant symptoms that caused the patients to seek medical care were nasal obstruction (11 cases); difficulty in deglutination (2 cases); epistaxis (2 cases), and hoarseness (2 cases). In the instances in which hoarseness was the chief complaint, the patients were found to have tumors in the pharynx as well as in the nasal or nasopharyngeal regions. Several patients complained of more than one of the foregoing symptoms. In summarizing the cases according to the anatomic location, we found that five occurred in the nose alone; one in the nasopharynx; while in two instances both these sites were involved. In some instances tumors were also present in the oropharynx, larynx, lymph nodes or other sites."

My case is a male, 37 years of age, whose chief symptoms were nasal obstruction, epistaxis, epiphora, purulent discharge from the left eye, and pain and tenderness over the left cheek.

I wish to report the following case of myeloma involving the lateral wall of the nose, the antrum, frontal sinus, ethmoid sinus and sphenoid sinus of the left side of the head.

REPORT OF A CASE.

CASE 1. A man, age 37, a native of Greece, was referred to my office by Dr. Sarafianos with the following history:

For the past two years the patient complained of a moderate nasal obstruction and mucopurulent discharge. Four months ago the patient felt pain in his left upper teeth. He went to a dentist who removed a few teeth. The pain was not relieved. He then went to a clinic where x-ray pictures were taken, and a diagnosis of chronic sinusitis of the left side was made. The left antrum was irrigated several times. After each irrigation free bleeding occurred and the nose

had to be packed to stop it. The pain in the left face became progressively worse. The patient's chief symptoms at this time were bleeding from the left nostril upon the slightest instrumentation, nasal obstruction (left), headache, epiphora with a pus discharge from the left eye, pain and tenderness over the left cheek.

On physical examination the patient was moderately well developed, though somewhat anemic looking. Except for the nasal and eye condition, the physical examination gave essentially negative results. There was a moderate drooping of the left upper lid. Epiphora and a purulent discharge of the left eye were present. On inspection of the nose, the septum was deviated to the right and in contact with the left middle turbinate. The left inferior turbinate was enlarged. Under the left middle turbinate a large grayish mass was present, soft to touch and bleeding freely upon manipulation.

The Wassermann test and urine examination gave negative results. Bence Jones was negative.

X-ray findings: August 24. There is a definite evidence of increased density over the left sphenoids, ethmoid, maxillary and frontal sinuses. The nature of the involvement suggests the presence of tumor infiltration.

X-ray findings: August 27. Lipiodol study: Lipiodol instilled into the left antrum. There is very little opaque dye in the left antrum. The only conclusion that one can draw is that there is an obstruction in the left maxillary sinus so that the fluid does not enter it.

BLOOD STUDY:

Differential:	
Polynuclear neutrophiles	72%
Polynuclear eosinophiles	1%
Small and large lymphocytes	24%
Monocytes	3%
Red cell study:	
Anisocytosis	0
Poikilocytosis	0
Macrocytosis	0
Oligochromasia	slight
Megaloblasts	0
Normoblasts	0
White cell count	6,850
Red cell count	4,210,000
Hemoglobin	81%

Section removed for biopsy. Diagnosis: Lymphosarcoma.

Preliminary operation, August 28th.

In view of the excessive bleeding obtained during slight instrumentation of the tumor mass, it was deemed advisable to ligate the external carotid on the left side. This was done with fascia lata.

Major operation:

External incision made over the region of the left maxillary bone, starting at the outer canthus of the eye, parallel to the lower lid to the region of the naso-

maxillary articulation, downward and curving to the center of the lip. The whole cheek was dissected free of the maxilla. The anterior wall of the maxilla was found greatly eroded with the tumor mass adherent to the underlying soft structures of the cheek. The maxilla was freed of its malar and nasal attachment. The palate bone was chiselled through. The soft palate was dissected free. The entire maxilla, lateral nasal wall and new growth was removed en masse. The ethmoidal and sphenoidal cells were completely exenterated. The left frontal sinus was thoroughly cleaned of a tumor like mass similar grossly to that found in the antrum. The mucous membrane within the mouth were sutured to best advantage. The cheek was returned to its normal position and sutured. The cavity was filled with vaselined gauze. Dressings were applied.

The patient was able to swallow liquids twenty-four hours after operation. He had an uneventful postoperative course and was discharged from the hospital two weeks after operation. At the present time he has a small defect in his cheek in the region of the inner canthus. This defect will be corrected in the near future by means of a plastic operation. He is having a dental prothesis made to fill in the defect left by the removal of the maxilla. He is comfortable, eats semi-solid food and can be understood in conversation.

Pathologic Report:

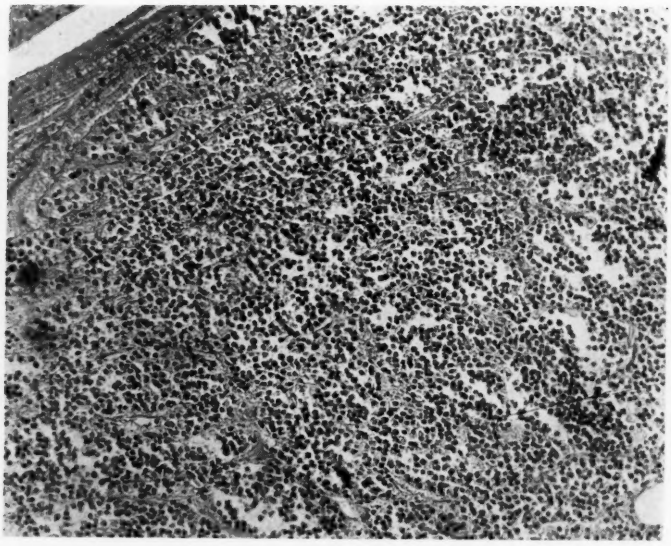
Material: tumor mass; bone, left maxilla.

Description, Gross:

The specimen consists of a portion of maxilla, gum and five teeth, measuring 6 x 3.5 x 3.5 cm.; portions of osseous tissue said to surround the maxillary sinus and several portions of moderately soft tissue measuring from 2 x 1.5 cm. to a globular mass measuring 5.5 x 4 x 3 cm., said to come from the cavity of the left maxillary sinus.

The large globular mass has a variegated yellow, gray, pink and red surface on one side. On the other side it is rough. The cut surface of this presents a lobulated yellow gray structure, surrounded and divided by delicate bands of gray tissue. In the center there is an area of hemorrhage measuring 2.5 x 1 cm. This structure is encapsulated in a thin, delicate gray capsule. One of the smaller portions of tissue resembles the mass just described. The rest are more or less flat portions of tissue resembling mucous membrane of the nasal cavity. A section of the larger globular mass is preserved for microscopic study; also a section of the smaller mass.

Accompanying the above described specimen is a mass of tissue measuring 1 x 1 x .7 cm. It is moderately firm and covered by a delicate gray membranous structure. The cut surface presents a finely lobulated pink and light brown structure. One portion is preserved for microscopic study.

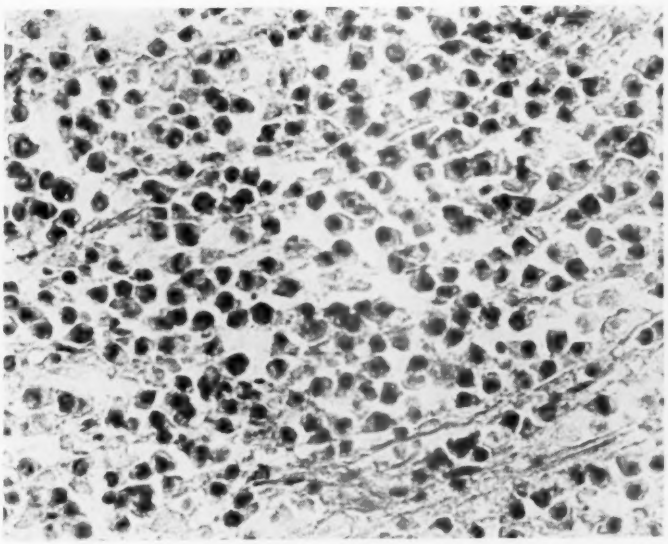


Plasmocytoma of maxillary sinus (200 x); portion of tumor showing distribution of plasma cells. (Author's case.)

Microscopic:

Sheet of small round cells with deep staining nuclei eccentrically placed in a scant amount of cytoplasm, are imbedded in a fibrillar, delicate ground substance with minute capillaries. Areas of necrosis are frequent. In places bands of fibrous connective tissue with a few cells separate the sheet of cells. Large thin walled lumina are frequent in some areas. In other parts, extensive areas of necrosis and hemorrhage are seen. Preparations from several other parts of the growth present much the same structure. In places the septa separating the sheets of cells are speckled with nuclear débris. A part of one surface of the preparations is covered with pseudostratified cylindrical epithelium, resting upon a band of hyalinizing edematous fibrous connective tissue.

The other microscopic preparation is a lymph node. The capsule is slightly thickened in places. There is a slight overgrowth of the germinal centers of the lymph follicles. There is a slight increase of the connective tissue of the septa, some proliferation of the sinus cells, and a dense infiltration with small round cells of all the sinusoids and septa.



Plasmocytoma of the maxillary sinus (800 x); portion of tumor showing cellular structure. (Author's case.)

Preparation stained by the May-Greenwald and by the Unna Poppenheim methods supply no additional information.

Diagnosis: Portion of maxilla (left), myeloma of, plasmocytic; lymph nodulitis, chronic.

COMMENT.

The question of the etiologic factor of the plasmocytoma still remains to be determined. Some authors regard this lesion a true neoplasm, while others think of this growth as a granuloma.

Blacklock and Macartney,[30] in reporting their case, argue that the fact that no organisms were present in the substance of the tissue especially stained for bacteria, is a point in favor of neoplasm rather than granuloma. They believe the tumor may be simple or malignant. "Apparently as with other tumors of mesoblastic origin, there are transitions from the simple to the malignant, and the classification suggested by Facchini and Scales[11] might be usefully adopted, the term plasmocytoma being reserved for benign growths and plasmosarcoma for malignant tumors."

Pollock[34] states that the classification of the plasmocytoma of the nose and nasopharynx is not certain, but he believes that they

SUMMARY OF CASES.

AUTHOR	DATE	AGE	SEX	SYMPTOMS DURATION ETC	LOCATION
Schridde[4]	1905	40	M	Not given	None
von Werdt[5]	1911	69	M	Difficulty in swallowing and nasal speech; growth excised and wound healed quickly with no recurrence.	Posterior surface of uvula.
Vogt[6]	1912	20	M	Sudden onset of hoarseness; pain in neck; patient died before operation; necropsy done.	Uvula, pharynx, tonsils, epiglottis, larynx, cervical, axillary and inguinal lymph nodes.
Kusunoki[7] and Frank Frank	1913	44	M	Lump size of pea at angle of left jaw; two years later tumor in nasopharynx with respiratory difficulty; epistaxis with loss of weight; died four years after onset, showing swelling of upper part of thigh.	Nasopharynx, left side of neck.
Wachter[9]	1913	48	F	Onset twenty years previous with slight hoarseness and pain in neck; operation; recurred locally; again removed; recurrence.	Floor of right nasal fossa and inferior turbinate; floor of left nasal fossa; velum above uvula; left false cord.
Rogers[10]	1920	42	M	Severe attacks of epistaxis for a period of a year, nasal obstruction; growth removed; uneventful recovery.	Inferior border of cushion of eustachian tube, and adjacent surface of soft palate.
Faccini[11] and Scalas	1925	47	F	For two years nasal obstruction; operation; no recurrence after a year.	Right nasal cavity from posterior portion of septum.
Rössle[12]	1926	54	M	Nasal obstruction; extirpation of tumor filling nasopharynx; death several days later; necropsy.	Tumor grown through clivus, filled sphenoidal sinus; roof of pharynx involving upper part of left nose; post. surface of soft palate; mucosa of left antrum, cervical nodes, sternum ribs, skull.
Hückel[13]	1927	46	M	Difficulty in nose breathing for six months with almost complete nasal obstruction.	Nasopharynx and mucosa of posterior edge of wing of the vomer.
Pallestrini[14]	1927	43	M	Nasal obstruction; nasal discharge; frontal headaches; pain in region of distribution of first and second divisions of the fifth nerve, right.	The inferior half of the right lateral wall of the nose.
Bronzini[15]	1928	53	M	Nasal obstruction with mucopurulent discharge; frequent sneezing.	Inferior turbinate, right.
Borri[16]	1928	56	M	Difficulty in eating solid food; nasal occlusion; operation, recurred locally; operation; recurrence; disappearance following antisyphilitic treatment; Wassermann test positive.	Left nasal fossa; right palatine tonsil; posterior pharyngeal wall.

Claiborn[1] Ferris	1930	34	M	Nasal obstruction; tumor removed; no recurrence.	Right nasal fossa filling entire interturbinate space.
Claiborn[1] Ferris	1930	60	M	Obstruction to right nose of two years' duration; nasal discharge; small growth removed; considerable improvement for short time; recurrence of tumor; tumor again removed; on recurrence to date.	Right nasal fossa extending from the middle meatus over the middle turbinate; fossa of Rosenmuller.
Blacklock[30] Macartney	1929	64	M	Hoarseness, difficulty in clearing throat; bleeding from throat; recurrences in original site and fresh sites; recurrences four times.	Larynx; nasopharynx and posterior wall of nasopharynx, five growths.
Cooper[35]	1927	45	F	Pain and nose bleed. Diagnosis: Plasmacytoma of nasal bone.	Swelling of left side of nose; ulceration of upper portion of left nares; tumor tightly adherent to the left lateral wall of the nasal and maxillary bones. Patient treated with radium; lost track of patient.
Cooper[35]	1933	68	M	Burning sensation and swelling of left side of face; frontal and temporal headache, lacrimation; epistaxis; lost fourteen pounds.	Firm, nonfluctuant mass over left cheek, narrowed palpebral fissure; left anterior nares completely obstructed, with impingement of turbinates against septum[8] left posterior nares showed a portion of the tumor mass protruding into the nasopharynx.
Pollock[34]	1933	70	F	Nasal obstruction, mostly in the right nostril; post nasal dripping.	Large polyp under right inferior turbinate; septum deviated to left; posterior rhinoscopy reveals large mass in nasopharynx attached to septum on left side in region of vomer.
Pollock[34]	1933	39	M	Headache, nausea, projectile vomiting; paralysis of right side of face; attacks of unconsciousness; frontal headaches; diplopia; bleeding from throat; death.	Small granular mass just lateral to right eustachian tube orifice; bled easily on pressure; postmortem showed multiple myeloma of sternum, skull, liver and sphenoid bone.
Saarni[33]	1933	39	M	Mucus discharge from nose, obstructed breathing; heavy feeling in head.	Slight deviation of septum to right; mucous membrane red and bleeding; tumor in left nasal fossa; posterior rhinoscopy revealed both nasal fossa full of tumor; metastasis to ribs.
von Iterson[37]	1934	68	M	Discharge and obstruction from left side of nose.	Tumor, reddish and slightly mobile in left nasal fossa; posterior rhinoscopy revealed tumor obstructing the left choana; treated by x-ray.
Blumenfeld, L. (author's case)		37	M	Nasal obstruction; epistaxis; epiphora; purulent discharge from left eye; pale and tenderness over left cheek; headache.	Tumor in left nares impinging left middle turbinate against septum; tumor infiltrated the antrum, frontal ethmoid and sphenoid sinuses on left side. side.

are of true neoplastic origin. He also states that in pathologic processes of the nose and throat plasma cells are frequently found in relation to chronic inflammatory conditions, including syphilis and tuberculosis.

Cooper[35] is of the opinion that solitary plasmocytomata and multiple myelomas belong to the general classification of malignant lymphoma, and while histologically identical, clinically they represent different aspects.

Claiborn and Ferris[1] state that plasma cell tumors of the nose and nasopharynx are probably much more frequent than the specific literature would lead one to believe. Undoubtedly many growths clinically diagnosed as nasopharyngeal polyps and not studied microscopically have a predominant plasma cell structure. Accumulations of plasma cells may accompany syphilitic, tuberculous and other inflammatory processes to such a degree as to simulate true neoplasms.

Jackson, Parker and Bethea[40] reported several cases of myeloma with nasopharyngeal involvement. They believe that multiple myeloma should be classed among the malignant lymphomas.

Rosenwasser[36] was of the opinion that plasma cells are derivatives of lymphoblasts, and that the plasmacytoma is a lymphoblastoma with more or less extensive cellular transformation to the plasma cell type. This places the plasmocytoma near the lymphosarcoma of the nasopharynx.

I wish to express my appreciation to Dr. E. L. Berger, for his advice and assistance in the operative and postoperative care of this patient.

SUMMARY.

1. The etiologic nature of plasma cell tumors of the nose and nasopharynx is still to be determined.

2. I have reported twenty cases of plasmocytoma of the nose and nasopharynx. Fourteen of these cases were abstracted by Claiborn and Ferris up to 1930. Seven cases were abstracted by the author. These cases were reported between the years of 1930 to 1935. The author has also reported his own case in detail.

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XXVI.

THE PHARYNGEAL LYMPHATICS AS A FOCUS OF INFECTION.*

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Our admired and beloved Sir William Osler said to his students on one occasion, "The knowledge that a man uses, is the only real knowledge, the only knowledge which has life and growth in it, and converts itself into practical power," and as the master has so aptly said, we must be prepared with an open mind to think and consider carefully any practical or working knowledge which new theories may present for our perusal; hence in presenting this subject, I wish to call your attention to the fact that I recognize,

First: That any involvement of the lymphoid follicles or glands under discussion very rarely, if ever, is a primary focus of infection.

Second: That as a secondary focus it is a definite factor and source for the distribution of infections, and has long been overlooked or unrecognized as such.

Third: The elimination of this focus would aid in preventing infections which originate in the adenoids, tonsils, teeth, sinuses, etc., from being continued indefinitely.

My intense interest in this individual field has been aroused by the observation that although it is common knowledge that multiple foci of infection or sources of reinfection exist and that we should use every means in our power to isolate and eliminate all of them, this area has too frequently been overlooked.

Perhaps the best means to draw attention to this overlooked focus is to include it in a brief anatomic résumé of the lymphoid structures and lymphatic drainage of the region with which we are concerned.

In its simplest form, the lymphatic drainage begins as a plexus of minute, closed, lymphatic capillaries, surrounding and interdigitating between the microscopic constituents of nearly all tissues. These capillaries join to form larger vessels which, in turn, empty into re-

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gional lymph nodes. Those groups with which we are particularly concerned are the retropharyngeal, the superior deep cervical and the inferior deep cervical groups. The channels from these groups continue downward to enter the general circulation by way of the right lymphatic duct, and on the left, the thoracic duct. It is known that they may have direct connection with the axillary nodes and, suspected on very suggestive evidence, that they may also have a more or less direct connection with the tracheo-bronchial groups.

Returning to the lymphoid tissues of the pharynx, which the above system drains, we can list them arbitrarily in the order of their hitherto recognized importance and isolate a recognized but heretofore neglected area. They are, in order, (1) the tonsils, (2) the adenoids, (3) the so-called lingual tonsil, (4) the lymphoid tissue at the base of the tongue (these four forming the so-called Waldeyer's ring, (5) the pharyngeal tonsil, (6) the lymphoid tissue at the lower end of the eustachian tube and lastly, the islands of lymphoid tissue, more technically called follicles, interspersed throughout the surface of the pharyngeal mucosa, any of which may be hypertrophied and harbor infection. Of these, a certain group in the lateral walls, on either side of the nasopharynx and pharynx, are of particular importance in certain subjects. They appear as a string of follicles extending in a vertical chain from just below the fossa of Rosenmüller above, to a point below and behind the junction of the posterior pillar with the pharynx at their lower extremity. Whether due to their location, contiguity of diseased tissue, the fact that they are more or less constantly bathed by drainage from above, or their anatomic structure, this particular group of lymphoid follicles may and do retain infection over long periods of time, and are a potential focus which has long been overlooked and must be considered when searching for obscure sources of infection. These structures drain in exactly the same manner as similar tissues in this vicinity, i. e., into the retropharyngeal nodes and superior and inferior deep cervical nodes and the materials absorbed therefrom enter the general circulation by way of the lesser circulation through the lungs in exactly the same manner as above described.

From the above, one can readily conceive and realize the importance that should be attached to the lymphoid tissue in the area under discussion, and particularly to the chain of follicles in the lateral walls, as a contributing factor to many systemic involvements where the source of further supply of infection has not been detected. With the fact well founded that infection is disseminated by two principal routes: First, direct from the blood stream or as an embolic

process; second, by the lymphatics, lymph nodes and infected lymph, and in all such infections which usually are of a chronic or low grade type, develop insidiously and progress slowly, producing symptoms of either a local or systemic nature, we must examine and observe these pharyngeal lymph follicles very carefully, especially when all of the usual foci in the throat, sinuses and mouth have been eradicated or eliminated by repeated examinations.

Statistics show that in about 37 per cent of the cases of focal infection, the site is unknown—the essayist wonders if the infected lymphoid follicles in the area under discussion might account for some of these unrecognized cases. Possibly some of the painful throats, the so-called neuralgic or rheumatic type, or some of the low grade continuous fevers which have taxed our diagnostic ability, may also be accounted for by infection harbored in this lymphoid tissue. We must not be unmindful of the fact that the speed with which results are obtained following the elimination of this focus, just as in any other focus, depends somewhat upon the virulence of the infection harbored and disseminated and the focus being removed too late to affect beneficially in a short time a firmly seated pathologic process. These lymph follic'·s, in such instances, are merely acting as a station or reservoir, harboring infection for metastasis to other tissues in the body. However, as long as they continue to exist, harbor and disseminate an infection or its toxins, so in the same proportion will the pathologic process continue to exist and progress.

Time does not permit of the rehearsal of case histories. Suffice it to say that many systemic involvements, such as infectious neuritis, acute and chronic infectious polyarthritis, iritis, keratitis, myositis, pericarditis, adenitis and many others, may and can be prolonged by a low grade persistent infection in these lymph follicles, while on the other hand the elimination of this focus has in a great many instances obtained very pleasing results.

Consequently, in our examination of a case of any of the above infections processes, let us include the lymph follicles under discussion in our comprehensive analysis as a potential possibility.

Probably some well conducted research work covering a large series of very complete sensitivity tests on the throats of these patients, especially those who present a history of having had scarlet fever, may elicit some very valuable information.

It is interesting to note that these hypertrophied and infected lymph follicles in the pharyngeal walls, so frequently referred to as a granular pharynx, have received so little attention in the literature,

except in a very casual way, with no emphasis on the fact that they can be and are acting as a secondary focus and continue to distribute infection which has originated elsewhere. It also is an intensely interesting fact that so little thought and study have been given to the eradication of this focus. Several noted authors, such as Coates, Hutchings, Beatty, Cecil, Dunlap, Uffenorde and others have discussed this subject in a very limited way but have offered no solution of this very troublesome problem other than the complete removal of the original foci, leaving behind this focus to continue the propagation of the original infection.

Many forms of treatment have been instituted in the past for the elimination of the hypertrophied lymphoid follicles in the pharynx, such as applications of various strengths of silver nitrate, chromic acid, trichloracetic acid, etc., and in recent years, diathermy, radium and x-ray have been used with fair results. Of these, x-ray is the outstanding one and in the hands of a few men has given very satisfactory results.

The author's favorite method and best results have been obtained by treating this focus of infection the same as we would, were it located any place else, that is, by a very careful dissection, removing part or all of this chain of lymphoid follicles along the lateral wall, as well as the destruction of any other hypertrophied and infected lymphoid tissue in the pharyngeal mucosa. Of course, it goes without saying that this can and should be done at the time of any other surgery on the throat, whether under general or local anesthesia.

As an alternative to the above, especially where a general anesthetic is not possible, electro-coagulation may be used quite successfully, if not used too radically, and with great care and patience. The advantage of this latter procedure is, first, that it can be and is done under local anesthesia, the second, that it does not require hospitalization and usually the patient can continue at their work with very little inconvenience. However, this method requires much time, for the reason that the operator must destroy only a few of these follicles at a time and wait a considerable period of time for the reaction to completely subside before proceeding. I have used the last two mentioned methods on a great many patients over quite some period of time and have obtained, in the majority of cases, very gratifying results.

Diet, hygiene and general measures to improve the resistance of the patient naturally are essential, as in any infectious process; however, they have purposely not been discussed here.

In view of the fact that recognition and treatment of focal infection has in recent years come so prominently into the foreground as a causative factor in many of the above mentioned pathologic processes which are the result of chronic infections, I have presented this very interesting subject for your consideration and approval, with the hope that it will eventually prove to be a definite clinical entity, and through your constructive criticism we may stimulate more careful and thorough examination, observation, research and treatment of this frequently overlooked area as a focus of infection.

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XXVII.

EXTENSIVE BILATERAL SUBDURAL ABSCESS. MICROSCOPIC STUDY OF THE MENINGES AND BRAIN: REPORT OF A CASE.*

WILLIAM R. GERAGHTY, M.D.,

BALTIMORE.

The combative and protective powers of the leptomeninges are so well demonstrated in the following case, where intracranial infection was of long standing, that it seems worthy of report and comment. Observation is also made of the associated and secondary changes in the brain. The origin of this subdural infection was a suppurative left frontal sinusitis; however, there was no gross evidence of disease in the posterior bony wall of the sinus. The chronologic order in the production of the intracranial complication is also shown, together with an explanation of the mode of infection.

REPORT OF A CASE.

CASE 1. *History and Course.*—H. J., white male, aged 48, entered the Presbyterian Eye and Ear Hospital in the service of Dr. Harry Slack on November 16, 1931, complaining of purulent discharge from the throat and nose and left frontal headache, which was aggravated by jarring or stooping. This patient had been under medical care since December 16, 1930, when he first came to the Dispensary. This was his fourth admission to the hospital. Previously, on January 15, 1931, he had polyps removed from the left nares and a left ethmoidectomy; February 12, 1931, a radical operation on the left maxillary antrum, and October 22, 1931, the left posterior turbinates were removed and a left ethmoidectomy was again performed. This last operation was intended to increase drainage and relieve the symptoms caused by infection in the left frontal sinus. On the x-ray this sinus was cloudy. The persistence of the complaint noted above indicated the procedure was ineffectual and a radical operation of the left frontal sinus was advised and performed on November 24, 1931. Following this, there was a considerable purulent discharge, and his condition was satisfactory and afebrile for eight days. On the eighth day following the operation he complained of throbbing pain over the left frontal region, and there was tenderness and swelling of the left upper eyelid followed by swelling of the right eyelids, with a temperature of 100 degrees F. The temperature ranged between 99 and 101 degrees F. for three days, with a pulse of 90 and respiration of 24 per minute, and continuous throbbing pain in the frontal region. He then developed marked tenderness over the inner roof of the right orbit. The leucocyte count was 18,600 per cu. mm., with 89 per cent polymorphonuclear neutrophiles.

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It was suspected that the right ethmoid cells were involved, but exposure of these cells proved otherwise. During the following week the severe left frontal headache persisted and the patient became weak and listless. The tenderness over the right frontal bone and right orbit became exquisite and the edema increased. The eye grounds were normal. Purulent right frontal sinusitis and osteomyelitis of the frontal bone were thought present; extradural abscess was suspected. On December 22, 1931, the left frontal sinus was again opened through the former incision and purulent and polypoid material curetted out, but no exposed dura was found. As a small perforation in the frontonasal region was found leading into the right frontal sinus, the anterior wall of this sinus was removed and purulent and necrotic polypoid material curetted out. No exposed dura was found in this sinus, but an osteoperiostitis of the anterior surface of the frontal bone was observed. On December 25, 1931, he complained of nausea and pain in the back of the neck, his temperature rose to 102 degrees F., and the next day there was cervical rigidity and a suggestive Kernig was present. A diagnosis of meningitis was made and lumbar puncture performed. Cloudy fluid under pressure was obtained, with a cell count of 700 per cu. mm. Culture of this fluid on blood-agar plates was negative. Three days later his temperature rose to 103.4 degrees F., pulse 90 per minute, he vomited several times, became delirious, had a generalized convulsion, and became unconscious. He remained in this comatose state with temperature varying from 102 to 104 degrees F., and a rapid, weak pulse till 9:00 P. M., January 2, 1932, when he died.

NECROPSY.

In stripping the scalp forward a large amount of greenish pus welled up from the subcutaneous area in the region of the left zygoma. The external surface of the frontal bone showed the denuded roughened surface characteristic of osteoperiostitis and operative wounds of both frontal sinuses. When the dura was incised over the frontal part of the hemispheres pus poured out in a small stream. In removing the brain there was a small hole in the left frontal pole that was exuding pus, and this area was adherent to the covering dura. The dura was stripped down from the inner surface of the frontal bone, and no defect could be seen in the posterior wall of either frontal sinus. There was no defect in the floor of the skull. The ethmoid sinuses were opened and the left posterior contained pus. The sphenoid sinuses were opened and the left also contained pus.

After formalin fixation, examination of the dura disclosed a thick, light green, purulent exudate on the ventral part of its internal surface, but macroscopically the external surface appeared clear. The exudate covered the left side of the falx as far back as the tentorium; the right surface of the falx was free except at its most anterior part. The tentorium was not involved. The longitudinal sinus was patent throughout. The brain, viewed from the rostral surface, presented deformities on both hemispheres (Fig. 1). On the right, extending from the middle of the frontal lobe back to the Rolandic fissure, and from the median fissure laterally, there was a saucer-like depression

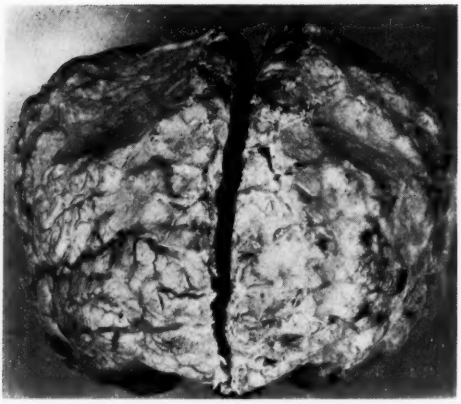

Fig. 1. Fibrinous exudate and deformity of both hemispheres caused by a subdural abscess.

.5 cm. deep in its center, and measuring 5½ by 4 cm. Over the left hemisphere there were three similarly depressed areas. The first, extending from near the median fissure outward and from the middle of the frontal lobe backward to the postcentral convolution, measured 5 to 3 cm. The second lay immediately inferior to this on the lateral aspect of the frontal lobe, over the operculum, and measured 2½ by 2 cm. The third involved the region of the fissure of Sylvius, the concavity being formed partly by the temporal and partly by the frontal lobes, its dimensions being 2½ by 2½ cm.

These depressed areas were covered with a velvety, grayish exudate, and the flattening of the convolutions gave the concavities a smooth surface. The underlying veins were smaller than over the rest of the cortex. All the pial veins posterior to these concave areas were greatly dilated but no exudate was present. The infection localized in the regions just mentioned and over the frontal poles. Both the latter were covered with exudate, and in addition there was an area of softening 1 cm. in diameter in the left. At autopsy pus was seen exuding from this.

The mesial aspect of the left frontal lobe was covered with exudate as far back as the anterior parietal region. On the right the mesial aspect of the frontal lobe showed the exudate present only for a distance of 2 cm. caudally from the frontal pole. In depth it did not go below the gyrus cinguli on either lobe. Viewed from the

base, the left frontal lobe was distorted, being shorter and broader than the right. Purulent exudate covered both frontal lobes back almost to the optic chiasm. On the left is extended into the Sylvian fissure and over the temporal lobe. The interpeduncular space, pons, medulla and cerebellum were uninvolved. The cisternæ pontis and cerebellomedullaris and the cerebellopontine areas did not show any exudate.

Frontal cuts were made and showed a small area of softening 1 cm. in diameter in the subcortex of the inferior gyrus of the left frontal lobe. No exudate was seen in any of the ventricles. The aqueduct was patent. Nothing abnormal was seen on transverse section of the cerebellum.

Smears made from the exudate over the cerebrum and inner surface of the dura showed gram positive long and short chain streptococci.

MICROSCOPIC ANATOMY.

The histologic observations are made from a study of blocks of tissue taken at various regions: all lobes of each hemisphere, including cortex and subcortex, basal ganglia and corpus striatum, pons, medulla, cerebellar cortex and centrum, ependymal lining and subependymal tissue in the ventricles, and from the superior and inferior longitudinal sinus and dura mater.

Sections from the left frontal lobe show a layer of fibrin superimposed on the piarachnoid, and the uppermost part of this layer contains many polymorphonuclear leucocytes. Beneath this the thickened mesh of the piarachnoid is relatively free of cells, only on occasional round cell being present. However, there is a perivascular round cell invasion of the pial vessels (Fig. 2). The mesothelial cells of the pia and arachnoid show degenerative changes. There is considerable perivascular connective tissue proliferation, fibers streaming in all directions from around even the smallest of the pial vessels. Phagocytic cells are scattered loosely in the mesh down in the sulci. There are vacuolated macrophages, gitter cells, round cells, plasma cells and fibroblasts present.

Beneath the pia there is no proliferative reaction on the part of the glia of the marginal zones, though the cortical blood vessels show edema and proliferation in their adventitial coat. There are a number of small cortical perivascular hemorrhages (Fig. 3). Minute areas of necrosis containing polymorphonuclear cells are also present in the cortex (Fig. 4). There are small hemorrhages in the molecular layer

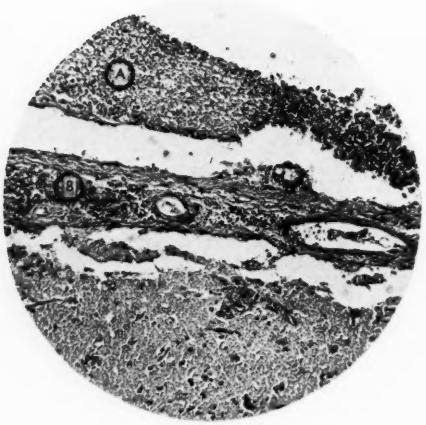

Fig. 2. *A.* Superimposed fibrinous exudate. *B.* Thickened meninges with perivascular round cell infiltration. Hematoxylin and eosin stain; x 150.

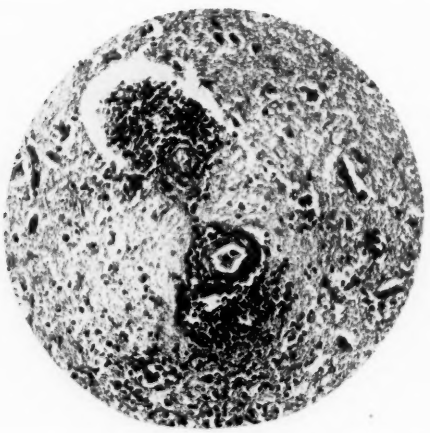

Fig. 3. Perivascular hemorrhages in the cortex beneath the subdural abscess. Hematoxylin and eosin stain; x 300.

with detachment of the meninges from the cortex. The cortical gray matter seems equally involved over the gyri and down in the sulci. Many cells of the cortex in these sections are preserved in outline only, being demonstrable as a homogeneous pale smudge; others are vacuolated, the nucleus being invisible, and pericellular edema is universal. The underlying white matter presents an edematous appearance. With MacCallum's stain, gram positive cocci in clumps are found in the pus on the meninges, but none are seen within the piarachnoid.

The area of softening shows a cavity with irregular, ragged outline, containing particles of necrotic tissue, clumps of cocci and many polymorphonuclear leucocytes. Extending back from the margins of the cavity the nerve tissue for a variable distance shows edema and granular degeneration with small blood vessels partially or completely thrombosed or hyalinized. Peripheral to this there is a marked invasion of small glia cells and a few small circumscribed hemorrhages. Beyond, approaching the less involved nerve tissue, some vessels present perivascular round cell infiltration, while others are surrounded by islands of fairly well preserved nerve tissue, beyond which there is circular rarefaction. Many compound granular corpuscles are found within and bordering the area of infection. Regressive changes seen in the nerve cells still present vary from simple ischemic alteration to almost complete destruction.

Over the right frontal lobe the meninges are greatly thickened by a deposit of fibrinous strands and covered by a granular layer of fibrin containing polymorphonuclear leucocytes. The mesothelial cells appear normal. Phagocytes are seen in great numbers down in the sulci (Fig. 5). These cells show various changes, the outstanding involvement being vacuolization of the protoplasm due to the presence of lipoid globules. The nucleus in a number of cells has disappeared; in others it stains faintly, showing two or three minute nucleoli. Granular degeneration of the cytoplasm is also observed. Some cells contain two nuclei. Giant macrophages are numerous. Lymphocytes and large plasma cells constitute a great percentage of the cell infiltration, but there are no polymorphonuclear leucocytes present except in the layer of fibrin on the meninges. Many round cells are present in the intima pia. Perivascular connective tissue proliferation is less marked than in the sections from the left frontal lobe. The capillaries of the deeper layers of the cortex show swelling or proliferation of the endothelium and the arterioles have thickened walls. There are some partly thrombosed arterioles. Petechial hemorrhages are present in the marginal zone of the cortex. Re-

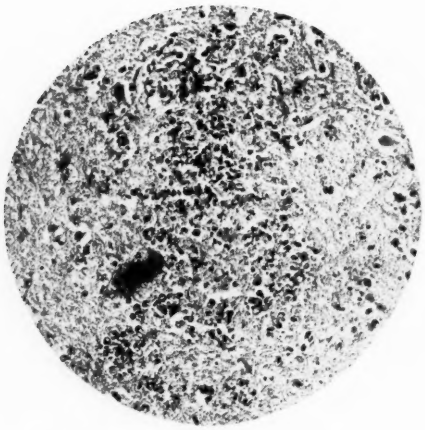

Fig. 4. Cortical focal necrosis. Polymorphonuclear leucocytes predominate; relatively few macrophages are seen. Hematoxylin and eosin stain; x 300.

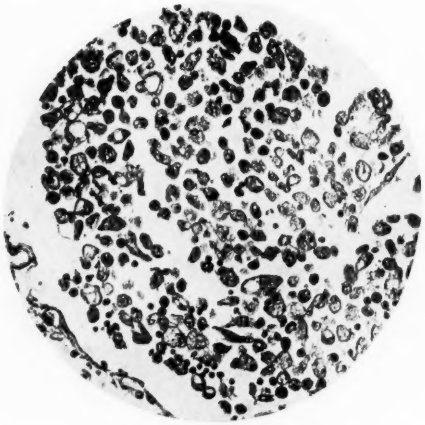

Fig. 5. Phagocyte cells: macrophages, lymphocytes, plasma cells, and fibroblasts. There are no polymorphonuclear leucocytes. Hematoxylin and eosin stain; x 300.

gressive changes are marked in the nerve cells, particularly in the second, third and fourth layers. The severity of the involvement lessens the farther one proceeds from the meninges toward the deeper cortical layers, the large pyramidal and polymorphic layer of cells being least affected. Pericellular edema is present. The underlying white matter in this area shows an absolute decrease in the demonstrable number of oligodendroglia cells.

The examination of the two temporal lobes presents different pictures. The findings in the left are similar to those in the left frontal. The right temporal lobe sections demonstrate a few phagocytic cells in the meninges with perivascular edema in the cortical and subcortical zones, and slight pericellular edema, though the nerve cells stain fairly well.

In the right parietal lobe there is an early leptomeningitis exhibited by a polymorphonuclear infiltration scattered throughout the piarachnoid and dilated, engorged vessels with leucocytes in their walls. Proliferation of the macroglia in the molecular area of the cortex is observed (Fig. 6). Edema is present in the cortex and to a lesser degree in the subcortex. In the left parietal lobe the vascular congestion and edema in the meninges, cortex and subcortical white matter are quite marked, but the leucocytic invasion of the meninges is not seen. Phagocytic cells, however, are present and show to a lesser extent the changes noted in those found over the frontal lobe.

The occipital lobes show a slight rarefaction of the molecular zone and pericellular and perivascular edema in the cortex and subjacent fiber tracts. The vessels of the meninges are congested, but other than an occasional histiocyte or lymphocyte there is no cell infiltrate present (Fig. 7).

The ependymal cells of the third, fourth and lateral ventricles are normal, though the subependymal glia is edematous. The epithelium of the choroid plexus does not present any abnormality.

No demonstrable parenchymal changes, other than a moderate degree of perivascular edema, are found in any of the basal ganglia or in the internal or external capsule.

In a section of the brain stem at the level of the trigeminal exit, immediately beneath the ventricle on each side of the midline, there are areas of perivascular hemorrhage similar to the cortical lesions. One of these on the right involves the substantia ferruginea. A number of these pigmented ganglion cells are disrupted, their pigment

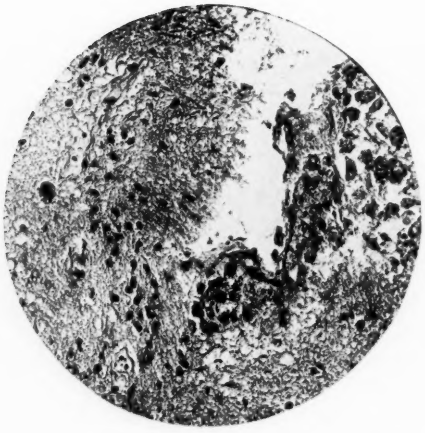

Fig. 6. Proliferation of macroglia in the marginal zone of the cortex. Early acute purulent leptomeningitis. Hematoxylin and eosin stain; x 300.

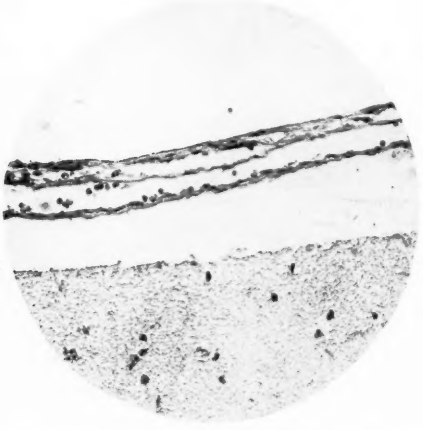

Fig. 7. Leptomeninges in the occipital region. Compare with Fig. 2. Hematoxylin and eosin stain; x 300.

collecting in dark granular masses in the adjacent edematous parenchyma and in the perivascular spaces and walls of the regional blood vessels. The meninges bordering the pons show a looseness of structure associated with edema, and the vessels are hyperemic, but there is no cellular infiltration. Sections caudal to this do not show any abnormalities.

Sections of the cortex and deeper structures of the cerebellum do not present any abnormalities.

There are no signs of infection in the superior or inferior longitudinal sinus. There is an exudate of fibrin (not visible macroscopically), containing leucocytes, on the external surface of the dura at the ventral extremity of the longitudinal sinus. The internal aspect of the dura shows, in places, invasion of the interstitial spaces by leucocytes and round cells. At other places collections of scavenger cells are seen. Foci of bacteria (cocci) are found in the dura and in the exudate on the external and internal surfaces.

COMMENT.

The original intracranial focus in this patient was a pachymeningitis with adhesion of the dura to the piarachnoid—a localized meningitis. The case is interesting because the meningitis, though involving a large part of the cortex of both cerebral hemispheres, remained localized for a sufficient time to permit the encysting of a large amount of pus (perhaps 250 cc.) between the dura and the piarachnoid. From the gross appearance of the brain (Fig. 1), one would suspect that the meningeal involvement was of long duration. Microscopically the greatly thickened piarachnoid, as shown in Fig. 2, when compared with the uninvolved meninges, as shown in Fig. 7, also indicates a long duration. The stormy clinical course during the thirty days preceding death could have been due to the extensive sinus infection or to the meningeal inflammation. Intracranial involvement was suspected three weeks before death, at which time the patient presented weakness and listlessness, suggesting increased intracranial pressure, and only late in the course did he present signs of meningeal irritation, for it was on the eighth day before death that postcervical rigidity and a suggestive Kernig were noted, and three days later he had a convulsion and was delirious.

The intracranial infection was undoubtedly an extension from the left frontal sinus. Destructive changes are microscopically more evident in the sections from the left frontal lobe than in any other. Leptomeningeal connective tissue proliferation is more abundant in

the sections from this lobe. Microscopic evidence of pathologic change is minimized proceeding from the frontal to the occipital pole, and the findings in the right parietal lobe are those of an early leptomeningitis. The extension from the left dural chamber to the right is explained by a spread of the infection in the dura crossing the falx. The right surface of the falx was practically free of exudate, and the findings do not indicate extension beneath or through it. The absence of visible disease of the bone in the posterior wall of the frontal sinus does not eliminate this sinus as the source of the intracranial infection. Zwillinger's experiments[1] demonstrated the communication between frontal sinus mucous membrane and the subdural space. Boenninghaus[2] considers that infection passes from the frontal sinus mucous membrane through the veins when the bone is macroscopically healthy.

Admitting the possibility of origin from the infection of the ethmoid or sphenoid sinus, through venous or lymphatic channels by direct extension, one would expect a primary basilar leptomeningitis, circumscribed or diffuse. The proximity of the chiasmatic part of the cisternæ basalis and its free communication with the other cisternæ would be more conducive to a rapidly spreading leptomeningitis. The lumbar puncture, eight days before death, showing a cell count of 700 and no organisms, contraindicates that the serious condition of the patient at this time was due to an extension of the inflammation in the meninges and leads to the conclusion that the late symptoms and death were due to the secondary encephalitis.

SUMMARY.

1. Subdural abscess may occur as a complication of suppurative sinusitis without demonstrable change in the bony wall of the sinus. Such an abscess secondary to frontal sinusitis is reported.

2. The primary intracranial complication in this case was a pachymeningitis followed by localized subdural abscess and encephalitis.

3. The piarachnoid forms a barrier of fibrin and connective tissue which localizes a subdural abscess and prevents the infection from spreading to the subarachnoid space.

4. Edema, hemorrhage and necrosis occurred in the brain immediately beneath the subdural abscess.

5. The spinal fluid findings of a pleocytosis and a sterile fluid indicate that the general subarachnoid space shows evidence of an

inflammatory reaction before the invasion of this space by micro-organisms.

N. E. Corner Lombard and Green Sts.

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XXVIII.

AN INTRAVENOUS ANESTHETIC IN TONSILLECTOMIES.*

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Evipal is chemically N-methylcyclohexenylmethyl barbituric acid. For a short surgical operation requiring a general anesthetic, it is comparatively safe, inexpensive and so compact that it can be carried in a vest pocket. It is of quick action, quicker than any other barbiturate, and it produces an almost complete anesthesia, with a notable lack of unpleasant after effects. By its use that great bugbear of general anesthesia called "psychic trauma" is commonly avoided.

The detoxication of this drug is due to its chemical disintegration in the organism, especially the liver. Observed effects on the circulation are inconsiderable; pulse continues normal; mucous membranes remain pink, though blood pressure may fall slightly; respiration is deep and regular. Metabolism, body temperature, alkali reserve and blood sugar level are not affected to any appreciable extent.

Dosage depends mainly upon the patient's weight, due consideration being given to his age, his general condition, and the type of operation. The maximum amount of a 10 per cent solution to be injected is six cubic centimeters for each hundred pounds of body weight, or 0.06 cc. per pound. A patient weighing 140 pounds, for example, should receive not more than 8.4 cc., or a child of 55 pounds not more than 3.3 cc. A total of 10 cc. should not be exceeded at one time. With children or elderly people the injection may be repeated, if anesthesia wears off before the operation is complete.

The entire dosage of this drug should not be injected at once, but at intervals of a few seconds; and the first 4 cc. for an adult, or less for a child, commonly gives an indication of the total amount to be used. If, in the case of an adult weighing 140 pounds, an injection of 4 cc. produces an immediate effect, then a total of 7½ cc. should be sufficient. As a safeguard, a laryngoscope should be at hand, and the glottic reflex observed. Complete loss of glottic reflex indicates

*Read before the Manhattan Eye, Ear, Nose and Throat Hospital Clinical Conference in New York City, 1935.

that more than enough of the drug has been injected, producing a deeper anesthesia than is necessary for a tonsil and adenoid operation.

Since there is no preliminary excitement, the pre-operative use of morphine sulphate is not necessary. The technic is the same as for other intravenous injections, and any vein will serve the purpose. Thus, in one case reported below, that of a four-year-old girl, a small vein on top of the foot was used.

The duration of anesthesia by this barbituric acid preparation averages from fifteen to twenty minutes. As contra-indications have not been entirely established, special care may be necessary in the presence of extensive injuries to the liver, because it is detoxified in this organ. Fatalities directly or indirectly associated with the drug are quite rare, and those having been reported were due to respiratory failure. No specific antidote is known; coramine has been suggested, and tried, but its effects have not yet been proven.

The committee on anesthesia of the British Medical Research Council reported favorably on the value of the drug. One author reports 10,000 patients treated without accident. In Germany another author mentions 25,000 cases with but one death.

The drug is especially suitable for short surgical operations of less than a half hour's duration. In eye, ear, nose and throat work, it could be used in certain myringotomies, peritonsillar abscesses, reopening mastoid wounds, abscesses of the neck, turbinectomies, intranasal antrum openings, and possibly cataracts, chalazions and some bronchoscopic cases.

The three following cases, selected from a much larger number of records, refer to tonsillectomies and adenoidectomies which have been done on patients ranging from the age of four years, weighing 50 pounds, to adults of 30 years, weighing 180 pounds.

REPORT OF CASES.

CASE 1.—A little girl, four years of age, weighing about 50 pounds. There was no definite reason for giving this drug, neither was there any contraindication. Her veins were so small and deep set that the only one accessible was on top of her left foot. A fine needle was used, with the usual preparation, and 3½ cc. of the drug injected. The speed with which it acted was amazing; complete relaxation was established before the needle was withdrawn. The tonsils and adenoids were removed in the usual way; a bleeding vessel on each side was ligated, though there was no undue tendency towards bleeding. The child reacted in about twenty-five minutes.

CASE 2.—A man, age thirty years, weighing 180 pounds. Eight cc. of the drug were injected into the cubical vein of the right forearm. As this did not seem

sufficient to relax him properly, the needle was kept in the vein, and the remaining 2 cc. injected, thus using the maximum of 10 cc. at one injection. The tonsils were removed in the usual manner by dissection, and there were no adenoids. It was not a particularly difficult case; however, it did clearly indicate that for the operator's convenience a mouth-gag should be in place before the drug is injected, as in adults it is sometimes troublesome to insert a mouth-gag after the strong muscles of the jaw have contracted. This patient required about thirty minutes to react after the operation.

CASE 3.—Of particular interest is the case of a boy, aged five, with an active tubercular condition in his lungs. This was indicated by an x-ray, taken on October 19, 1934, report of which read, "The chest shows a small shadow just below the right first rib, very suggestive of early tuberculosis." A physical examination revealed rales over both lower lobes, and a few moist rales under each scapula. A tuberculin test was positive in dilutions of 1:100, 000 and 1:10,000 and four plus in 1:1,000 dilution.

The child's tonsils were enlarged and infected; also the glands of the neck were involved. He had frequent colds, sore throats, and difficult breathing, a definite case in which an adenoidectomy and tonsillectomy was indicated. Obviously, he was too young for an operation under local anesthesia, aside from the difficulty of removing adenoids under local anesthesia. Because ether was definitely contraindicated, evipal was chosen. Four cc. were injected into a superficial vein of the right forearm. In less than fifteen seconds the patient was completely unconscious and relaxed. The adenoids were first removed in the usual way, and bleeding stopped by pressure. Both tonsils were removed with the snare, and a ligature placed on each side to stop bleeding. The only bad feature seemed to be that the tongue had a tendency to drop back into the throat, which necessitated constant traction. The glottic reflexes were abolished, indicating that more of the drug had been used than the case required.

In less than a half hour the child had fully reacted. He was discharged the following morning with a normal temperature. Three days later he was seen, but no ill effects were noted. Five months later he was returned to the clinic for examination, having gained several pounds in weight and improved in general condition. Another x-ray at this date reported: "This is a definite tubercular process but it seems to be well calcified."

In conclusion, since evipal proved to be an ideal anesthetic in this case, it suggests the hopeful question, why should not an anesthetic be perfected which would be easy to administer, quick to relax the patient, free from bad effects, and definite in action?

810 GURLEY BUILDING.

REFERENCE.

Winthrop Chemical Co., Inc.: Bulletin, Vol. 10, No. 1, 1936.

XXIX.

THE PHYSICAL CHEMISTRY OF VASOGRAPHY.*

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AND
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This article† continues the theoretical development of opaque x-ray solutions, and also some of the practical developments from the basic theory. Clinical cases were reported in the original article and will not be dealt with here. The whole idea upon which these studies were made revolved around the use of the x-ray in the diagnosis of vascular lesions by the injection of shadow casting solutions.

The importance of viscosity was apparent, but not understood, until experimental studies were completed to substantiate the material set out in this and the previous article.* Reducing the problem to mathematics makes it understandable, and at the same time offers much opportunity for research in a field that has not been covered in its application to clinical medicine.

When two streams of fluid are flowing in a tube or blood vessel they will mix together when that velocity of flow is reached, known as the critical velocity, wherein lamina flow breaks into turbulent flow. Turbulent flow is a disturbing factor in obtaining good x-ray pictures of a blood vessel by injecting a shadow casting fluid into the blood vessel. The blood itself has a viscosity and a critical velocity close to that of water, both being comparatively small in value.

These facts are shown and compared for two solutions for shadow casting at the temperatures usually used for best results. As may be readily seen, the higher the temperature, the smaller and more closely together are the viscosities of water, blood and the solutions.

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*From the Department of Experimental Medicine of the University of Kansas School of Medicine.

†This article is supplemental to the original paper, published under the title, "The Physiodynamics of Intravascular Fluid Injections for Roentgenographic Studies," in the Annals of Otology, Rhinology and Laryngology, Vol. 44, No. 2, June, 1935. Corrections of the mathematics in the previous article are included in this article.

The same is true for their critical velocities. On the graph (see Fig. 1) these critical velocities are plotted parallel to the viscosities, by choosing a scale of value ten times larger for the critical velocity, which may be done, since they are parallel or proportional functions of one another. This fact is further shown by the linear relationship, by plotting viscosities and critical velocities against one another. Straight lines result when these values are plotted, which mathematically expresses a direct proportional relationship. Both solutions contain the same amount of total solids, but different proportions of the different solids are used in the two solutions being studied.

Actual experiment and theory approximately coincide for the fluids when flowing separately in tubes. It yet remains to learn what will happen when mixing occurs, with blood at different rates of flow, in different sized vessels, for any set temperature. This work has not been completed. At present it appears that the new Lindbergh apparatus for simulating pulsating blood flow, would give results more nearly approaching the actual conditions found in the body, by experimenting in glass and gum rubber tubes.

Clinical study on dogs has established the fact that the optimum temperature is 40 to 60 degrees F. (5 to 16 degrees C.). The solutions injected must have a viscosity of 50 to 60 centipoises. The corresponding critical velocity is 640 to 1,000 centimeters per second in .5 cm., inside diameter tubes. These values are many times that of water at the same temperature, it being 21.5 to 28.6 cms./sec. at 60 and 40 degrees F. Blood at 98 degrees F has a critical velocity of 61 cms./sec. These facts are sufficient for practical application of x-ray shadow casting solution to enable any physician to make use of these solutions for this purpose.

The critical velocity and viscosity of blood are variable factors, depending on several factors that vary. This results from the fact that blood is a colloidal solution, involving the suspended blood cells and platelets. For this reason, only an average value, as declared by different authorities, has been assumed by the writers of this article.

Solutions of great viscosity are difficult to inject. However, such injections have been made successfully and produced good pictures. In fact, a glucose solution, made viscous by such a low temperature that small ice crystals could be seen to pass into the femoral vein of a dog, with no harmful effects at all.

This suggests the possibility of giving cold blood transfusions. However, this has not been tried. In so far as shock is concerned by

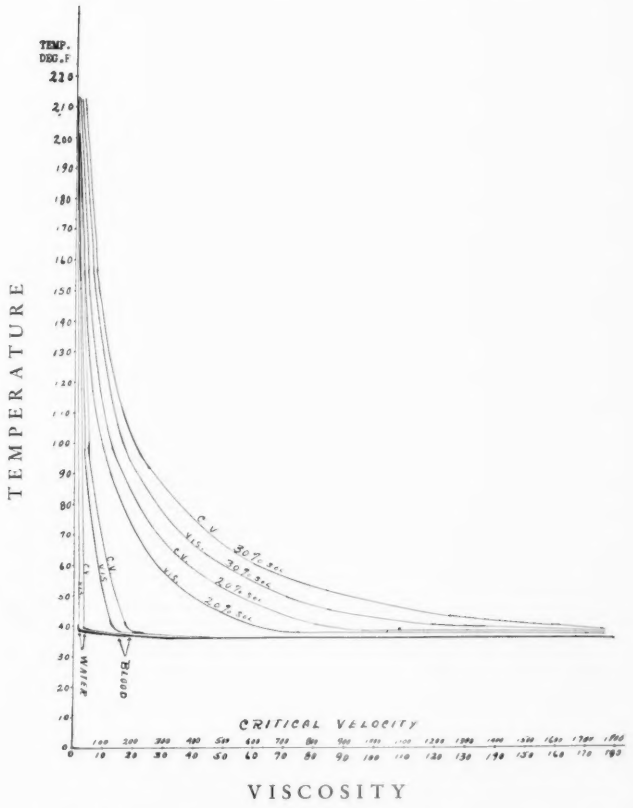

Fig. 1. The distance between points on this plot indicate the amount of increase in viscosity and critical velocity with temperature decrease.

Each succeeding 20° F increase in temperature results in a decreasingly small change in viscosity and critical velocity. As the higher temperatures are reached viscosities and critical velocities come much closer to one another. Apparently 50° to 60° F is the best temperature to inject the solution. The solutions and blood freeze below water. Freezing temperature, 32° F.

the injection, there is nothing to indicate the slightest objection to such a procedure. The possibility of blood clotting is still to be determined, which may or may not happen when hot and cold blood are mixed in the body, although we know that cool blood is the more difficult to coagulate. Blood will coagulate at temperatures around freezing. Alkalin solutions also prevent coagulation. The experimental studies made apparent the need for a cool fluid to be injected, because a higher viscosity was needed to produce the best

results, and at the same time not increase the amount of the solid materials in the solution.

It has been the practice among veterinarians for some time to inject serums and antitoxins cold. The absorption is slower and therefore results in a less disturbing reaction in the animal. The same may apply to the human.

The effect of cold solutions on the rate of hemolysis, and perhaps the total prevention of it, is not known, but there must be a vast slowing down of this physical change. This is deduced from the fact that low temperature decreases solubility of many substances, and that osmosis is slowed down by low temperature. Hemolysis is a result of endosmosis of water into the greater concentration of salt solution in the red blood cells, causing them to swell and burst. It has been found that hemolysis is not caused by sugar and salt solutions, but it is caused by ammonium salts and water in excess. Lecithin and cholesterol, which are part of the stroma of a cell, seem to prevent hemolysis unless they are soluble in the salts injected. Bile salts, soaps, ether, chloroform and amyl alcohol are solvents of these two, and will therefore penetrate the cell membrane and cause hemolysis. Schaffer, in 1915, states that glucose decreases autolysis of cells, and offers an explanation based on the protein sparing action of glucose.

All of these things point to the need of a compound in a solution to be used in blood vessel injections that will prevent destruction of the blood or tissue cells. Herein, most likely, lies the reason for unfavorable results following injections of many other shadow casting solutions into the blood stream. Even after passing into the other tissues, autolysis has resulted, and no doubt for the same reason. This is particularly noted in the lung tissues, where autolysis occurs when sodium iodide and similar irritating solutions that are injected into the veins, reach the lungs. The use of sugars similar to glucose prevents this when used with the proper salts for shadow casting. The particular advantage of a viscous solution is that fewer blood cells come into contact with the solution throughout its course up to the point of elimination in the kidneys. Elimination is hastened by the diuretic action of glucose or dextrose.

Another important factor in choosing a salt for shadow casting is to obtain a salt that will not pass through the blood cell membrane. The red cell walls are permeable to many dyes and not to others. Overton established this fact many years ago. He discovered that sulphonic acid dyes, which were soluble in water and not soluble in

TABLE I.—VISCOSITIES AND CRITICAL VELOCITIES.

	20% Iodine Comp. (contains 10.4% Iodine)			30% Iodine Comp. (contains 15.6% Iodine)		
Temperature	Density gms/cc	Absolute viscosity in centipoises	Critical velocity cms/sec	Density	Absolute viscosity	Critical velocity
5° C-41° F	1.3725	61.75	846	1.5215	132.2	1630
15.5° C-60° F	1.3667	28.5	392	1.5151	52.0	640
26.5° C-80° F	1.3618	16.6	229	1.5060	29.15	364
38° C-100° F	1.3548	9.155	127	1.5005	16.82	211
	Blood			Water		
Temperature	Density	Absolute viscosity	Critical velocity	Density	Absolute viscosity	Critical vel.
5° C-41° F	1.425	13.6	180	.999965	1.52	28.6
15.5° C-60° F	1.250	8.0	120	.9999023	1.138	21.5
26.5° C-80° F	1.175	5.0	80	.996649	.90	16.4
38° C-100° F	1.075	3.5	61.0	.992950	.685	12.9

The critical velocity for water at 68° F (20° C) in a two inch pipe is 1.8 cm/sec., which checks closely for the values found for a .5 cm. tube as given above. The smaller the tube, the greater will be the critical velocity. It will be seen that an injection of 30% solution at 60° F has 10.5 times the critical velocity of blood at 98° F. The absolute viscosity ratio is 15 times. It will be noted that an increase in density also increased the viscosity at the same temperature. The greater the density, the greater is the resistance to flow for any given temperature. Each of the iodine solutions contain a total of 70% solids. The viscosity of blood is a variable factor. Consequently, the critical velocity will be variable.

The above readings for viscosity were made with the Oswald apparatus.

oils and fats, did not penetrate the red cells and cause hemolysis. His work was done in the late 1890's.

This fact has been followed in preparing a solution for shadow casting that would be non-injurious to the patient.

The pH of the solution is also to be considered, since autolysis of tissue occurs least rapidly at pH 7.4 to 7.8 and most rapidly at pH 5 to 6, when enzyme activity becomes greatest.

The leucocytes are destroyed in media of high acidity corresponding to a pH value below 3.5, therefore neutral salts must be chosen. It is known that .1 per cent solution of sodium carbonate or bicarbonate do not cause hemolysis, whereas 1 to 2 per cent solution do cause it. Also neutral salts and excess carbon dioxide, and lowered temperature prevent blood coagulation. Other compounds, such as sodium citrate, oxalates and tartaric acid salts prevent coagulation.

Certain neucleoproteins, when injected, cause blood to coagulate, while hirudin prevents coagulation.

The use of glucose has the effect of so changing the electrical balances as to relieve pain and other harmful effects in that it serves as a dialectric.

In addition to these facts one main point about a viscous solution is that it has the effect of slowing down physical chemical changes which would be harmful if they occurred. Another point in favor of a viscous solution having high solid concentration is that it causes the cell fluids to flow out of the cell by osmotic action, which is not harmful, because a collapsed cell will return to its normal size by taking in fluid when it again comes in contact with blood free from the injected solution. A viscous solution appears to produce a more rapid diuresis, which is in no way harmful but may be a distinct advantage in that it hastens complete elimination. The reverse action destroys the cells and may be sufficient to cause serious disturbances. The exact point of osmotic equilibrium has not been determined, where osmotic action ceases or is at a minimum, but for clinical use it appears to be of no consequence when using concentrated glucose solution.

Another fact has been established with reference to shock of cold injections. The graph record of blood pressure, and pulse rate are so little changed from normal and in no way harmful to the patient when cold injections of viscous solutions are made to

the extent of a total of over one pint of solution injected in quantities up to 100 cc. at a time, at temperatures as low as 0° C. This study substantiates the assumption made and stated in the original paper regarding blood pressure changes.

This experimental data will be presented in a separate paper.

Another fact has been established with reference to shock produced by injections made into the peritoneal cavity, which were absorbed with no harmful effects and produced a good shadow. By taking one picture after another at the same locality, while the solution is being gradually absorbed and eliminated, gives shadow depths that differ enough to aid diagnosis by reading the series of pictures produced. A picture was made that showed the veins in the lungs, something which had not been done previously.

The facts and studies referred to in this and other articles, as well as unpublished data, have been the guiding principles used in preparing the solution of such composition as was found to give the proper viscosity and x-ray shadow casting capacity.

Many details as to the required time for making the exposure and the time after injection for taking the picture for different locations in the body are yet to be worked out. For injections in the vicinity of the desired picture the snap is made at the end or near the end of the injection period, and have always proved satisfactory.

CONCLUSIONS.

No definite conclusions are being drawn at this time because the writers feel that these studies are so far from the orthodox methods that they will warrant an extended amount of research by as many workers as can give time to the problems. This method suggests a means of determining rate of flow and many other related phenomena within the vascular bed.

CORRECTIONS IN "THE PHYSIODYNAMICS OF INTRAVASCULAR FLUID INJECTIONS FOR ROENTGENOGRAPHIC STUDIES"

Appearing in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, July, 1935

It is desired to correct mathematical mistakes that appear in the July (1935) issue of the ANNALS in the article by Dr. O. Jason Dixon on the Physiodynamics of Intra-Vascular Fluid Injections for Roentgenographic Studies. Proper corrections were not made in the galley proof. Therefore, the necessity of these corrections. On page 394, and paragraph 4, at the foot of the page, there is a mistake in the Fanning equation dp, equals $4fslu^2/2gd$ which is written here as it should be, with the (l) omitted. For some unknown reason, an extra (l) was put into this equa-

tion, and letter (p) used for the Greek symbol Rho. In the footnote on page 395 the symbol for Rho is given as (S), and is therfore used in the equation above.

In the last equation on page 394 the letter (u) has been used in place of Greek Mu. The symbol (y) has been used by the printers for Mu, therefore the correct equation becomes, dp, equals, $32ylu/gd.^2$

The footnote on page 395 should have appeared on page 394. While these corrections are being made a little more detailed explanation is given for those mathematically inclined. The value for (f) is a complicated value involving friction as may be seen in the test on Principals of Chem. Eng. by Walker, Lewis and McAdams.

Cardiograph readings showed no change of consequence in the blood pressure during these experiments.

Since this is true, the pressure drop in blood stream flow may be assumed constant with the injection, therefore equating the two equations for equal pressure difference dp, there results:

$4flsu^2/2gd = 32ylu/gd^2$, or $u^2/u = u = 32yl/gd^2 \times 2gd/4fsl = 16y/fsd$

Since $(16/f)$ in $(16y)/fsd$ reduces to 942, therefore $U = 942y/sd$.

In other words, everything cancels out until the value (U) for critical flow is obtained. The value may also be calculated as cms. per sec., as well as feet per sec., from the formula U equals $942y/sd$.

y = poise, s = density, d = dia. blood vessel, v = velocity, cm. or ft. per sec. The unit used must be consistent with the system used. It is much simpler to use the metric system of measures.

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The animal experiments and the viscosity readings were carried out in the Pharmacology Laboratory of Dr. Robert M. Isenberger, at the University of Kansas School of Medicine. We are also indebted to Mr. Mark Carroll, our technical assistant.

The x-ray studies were made available through the courtesy of the General Electric X-ray Corporation, who very kindly furnished a portable x-ray and all of the necessary equipment.

XXX.

PREVENTION OF POSTAURICULAR FISTULA BY PERIOSTEAL AND SUBCUTANEOUS TISSUE FLAPS.*

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DIXON, ILL.

A certain number of radical mastoids have the disagreeable complication of a postauricular fistula which should be closed. This dirty, discharging wound is carried by many people because they fear to undergo further surgery.

From the amount of literature on the various methods of repairing such a fistula one knows that it is not easy to get good results, as it is necessary to operate in a more or less infected area and usually there is a deep cavity to be filled in or bridged over. The etiologic factors producing postauricular fistula are:

1. Failure to remove enough of the facial ridge to allow dependent drainage.

2. Unusually deep cells at the tip.

3. Most important of all is the viability of the patient and his tissues (hygienic condition or debilitating diseases, such as scarlet fever, diabetes, tuberculosis, etc.)

Out of sixteen radical mastoids done in the past ten years in private practice, thirteen have dry epithelialized cavities, one died of meningitis after a primary skin graft, and two were so unfortunate as to have moist ears with postauricular fistula. One of these was an old scarlet fever ear and the other patient had diabetes. Both refused further surgery. But in an attempt to clean up some of the chronic otorrheas of inmates of the Dixon State Hospital for the feeble minded and epileptic, out of the first four patients operated upon three had persistent postauricular fistula. This additional disability made the care of the patient more expensive, enraged the relatives and necessitated further operative work, which is slow, tedious and only partially successful. Not one had a dry ear.

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*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Toronto, June, 1935.

The pathology of these mastoids could be classified according to Beck's[1] classification as either osteofibrosis or osteofibrosis fistular tracts and cholesteatomatous infiltration. There were no unusual complications.

The operations were done as near as possible after the teachings of Kerrison[2] with the facial canal well lowered. A Siebenmann flap was done on the ear canal which, according to Rae,[3] is the best.

The results, as have been before stated, were three postauricular fistula and the fourth has a patent tube from which mucus exudes. The fourth patient was a chronic epileptic and has not had a convulsion in the nine months since operation. It was absolutely necessary to do something to combat this series of postauricular fistulas, which was felt to be due to the poor hygiene of the patient. Although they have proper food, they do not get the same results from the operative procedure as do other patients. One patient recently admitted had previously submitted to twenty operations on his two ears and now he has postauricular fistula and foul ears. Our problem was the rearrangement of the postauricular tissues with view to the increased blood supply to the part. The literature was reviewed with reference to the various types of flaps and grafts to repair the fistula.

Straastma and Peer[4] repair the fistula by means of a free flap graft from the scalp. Eagleton,[5] during the war, used bone chips to close the mastoid wounds. He was the first to suggest that fat be employed. Hartwell[6] shows by experimentation that fat is of the greatest importance in the healing of surgical wounds. Watson[7] also feels that fat is a great factor in repair of the wound. Claus[8] uses bridging flaps to repair the wound. Ross[9] feels that the treatment of acute mastoiditis with x-ray is of great benefit. This has been considered, but not tried, as an adjunct before radical operation. Popper[10] uses small periosteal flaps after radical mastoid operations.

As for the mastoid itself, there are a great many different methods of skin grafting. Dench[11] reports in 1906, 193 cases, 131 cures, 6 deaths, and feels that it is the operator's choice, primary or secondary skin graft or none at all. Mosher,[12] in 1925, gives details of his excellent skin graft with the use of paraffin bucket. White, Jr.,[13] in 1928, reports cases, 57 per cent of which were cured following skin grafting, but he does not feel that the method is important. Botteman[14] warns against grafting over the tube. Day,[15] in 1934, reports fourteen radical operations all well, but he does not give the type of operation. All agree with Wittmaack[16] that cholesteatoma will only occur if there is an inadequate drainage of the exfoliated material.

Fig. 1. Shows regular mastoid incision with extended incision for flap.

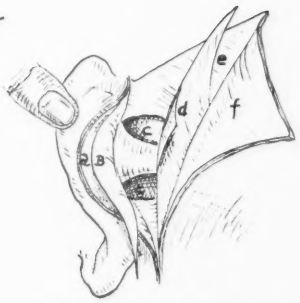

Fig. 2. *a*, Skin; *b*, Periosteal and subcutaneous tissue flaps; *c*, Mastoid cavity; *d*, Periosteal and subcutaneous tissue; *d*, Flaps; *e*, Bone; *f*, Skin.

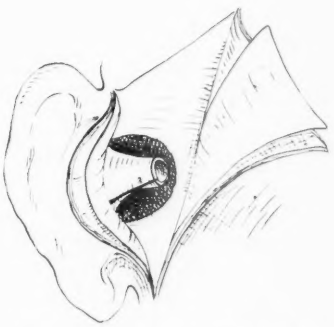

Fig. 3. *a* shows incision in lower margin of membrano-cartilaginous canal.

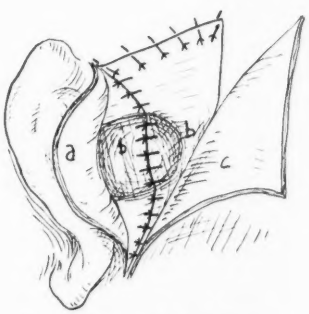

Fig. 4. Periosteal flaps sutured into place with chromic suture; *a*, skin; *b*, periosteal flaps; *c*, skin.

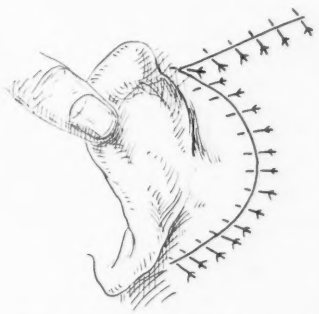

Fig. 5. Skin closed with vertical mattress silk worm gut sutures.

Almour[17] shows the significance of the squamous epithelium in the cause and repair of chronic middle ear disease and emphasizes the necessity of a large meatal opening by various plastics.

Following a study of these reports, a flap was sought that would permit closure of postauricular wounds, provide wide opening for drainage through the external auditory canal, and encourage growth of epithelial covering. To cover and fill the mastoid cavity and tip, the adjacent periosteum, together with a fatty subcutaneous tissue, was chosen.

A typical postauricular incision for the radical operation is made, then at the upper tip of this incision a new incision is made at the right angles, upward and backward, for a distance of about two inches, dividing all tissues down to the bone. This flap is elevated, the anterior flaps are elevated and pushed forward, and the membrane-cartilaginous meatus is completely separated from the posterior wall of the bony meatus. These two flaps give excellent exposures and most of the resultant scar is in the hair line. After the mastoidectomy has been completed, the margins are beveled carefully. The skin of the anterior flap is now dissected from the subcutaneous tissue and periosteum. This dissection is carried down into the membranous canal. A thin bladed knife is now introduced into the membranocartilaginous canal and, hugging the floor, an incision is made outward until it bisects the incision from behind. By hugging the floor the flap is now free, and it is used to form the roof, so there is no obstructing ledge below to interfere with free drainage. The periosteum and subcutaneous tissue is now freed from the skin in the posterior flap. The under flap is now freed from above downward to a distance about one and one-half inches behind the primary incision. This allows the graft to be moved forward and sutured by interrupted chromic sutures to the periosteal and fat graft from the anterior flap. Great care is taken to tie all bleeding vessels and to have the mastoid cavity free of blood and débris before the periosteal flaps are brought into place. The posterior auricular wound is then closed by vertical mattress silkworm-gut sutures. The membranocartilaginous flap is now held in place by packs. The after care is the same as for all mastoids.

One private patient with a chronic suppurative ear of nineteen years' duration and six institutional patients with chronic ear suppurations, varying from two to ten years' duration, have been operated upon using the above described flaps. The private patient had

a dry ear with a nice epithelialized cavity in forty-four days. Of the institutional patients, three have dry ears with good cavities; the other three are somewhat less foul than they were previous to operation. These are not wonderful results, but those that work with humanity of such poor hygiene and low tissue vitality know how difficult it is to get good results.

The contradindications of these flaps are facial nerve pathology and signs of intracranial complications.

The advantages of these flaps are:

1. The removal of a great deal of strain on the postauricular tissue.

2. The mastoid cavity is made much smaller.

3. A minimum of bare bone remains exposed after the operation.

4. The bone is covered by a tissue fashioned by nature to do so, and healing is more rapid owing to the profuse blood supply.

5. Liability of reinfection or of foreign introduced infection is very materially reduced.

6. The procedure is very simple and requires very little additional time.

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XXXI.

IMPROVED TECHNIC FOR SUBMUCOUS TURBINECTOMY.

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LOS ANGELES.

In an article published in 1930, Odeneal describes a submucous turbinectomy of the middle turbinate bones. At the close he says that he had never followed this procedure upon the inferior turbinate bones, but did not see why it could not be done when indicated. Although I cannot claim priority to the submucous turbinectomy, I wish to report three cases of submucous turbinectomy of the inferior turbinate bones by an improved technic.

To review briefly the fundamental anatomic and histologic details of the inferior turbinate bone: The inferior conchæ are two shell-like laminæ of bone, lying along the lower part of the lateral wall of the nasal cavity on either side. They are elongated and have curved borders separating a medial and a lateral surface. The superior border is the attached border. The inferior, or free border, with which we are concerned, is curved from before backwards and turned slightly laterally, is round and full. It is formed of bone which is deeply pitted and of a somewhat cellular character. The medial surface projects into the nasal cavity; convex from above downwards, and slightly rough and pitted. The anterior and posterior extremities are pointed.

The nasal mucous membrane is divided into two regions, the olfactory and the respiratory; the inferior conchæ are covered entirely by respiratory mucous membrane. It is thick and very vascular. It is adherent to the underlying perichondrium or periosteum. The mucous membrane has a stratified ciliated columnar epithelium. It rests upon a cribriform basement membrane. The ciliated cells extend the entire thickness of the epithelium. There are many mucus containing goblet cells. The glands are very numerous and largely of a mixed character.

The greater part of the nerve supply comes from the sphenopalatine ganglion by way of the anterior palatine nerve, which gives off the inferior postnasal nerve. This enters the posterior part of the inferior turbinate and subdivides in its substance, terminal filaments reaching the middle and anterior parts. The latter also derives part

of its nerve supply from the lateral nasal nerve, a branch of the anterior ethmoidal nerve. These nerves enter the inferior concha from the anterior superior part.

The blood supply is quite profuse. The posterior lateral nasal arteries, which are of considerable size, are branches of the sphenopalatine artery which in turn is a branch of the middle meningeal artery. The sphenopalatine artery, when it reaches the posterior lateral wall of the nose, divides into its terminal branches which supply the middle and inferior turbinates. The anterior ethmoid artery supplies the anterior portion of the inferior conchæ and anastomoses freely with the lateral nasal arteries. Consequently the operation may be attended with considerable hemorrhage. Bleeding may come from both sources, though but one artery may be injured.

The venous drainage follows the course of the arteries and, due to the cavernous character of the vessels, there may be considerable trouble from hemorrhage.

The respiratory function is threefold in character: (a) The outside air is raised or lowered to the body temperature, as the case may be, in passing over the mucous membrane of the nose. (b) The nasal mucous membrane, especially that of the inferior conchæ, contributes a certain amount of moisture, thus protecting the delicate air vesicles of the lungs from the varying humidity of the atmosphere. (c) The serum secreting glands of the inferior conchæ give off moisture which is rapidly taken up by the expanded air and carried to the lower respiratory tract.

The part of the mucous membrane which secretes the greater part of the serum is located chiefly along the inferior border and along the posterior end.

It can readily be seen that in such a structure various pathologic processes may be found.

In the early stage there is a distension of the venous sinuses or cavernous tissue of the turbinates. The secretions are heavier; that is, the mucoid element is increased, while the serous element may be decreased in quantity. To the patient there is an actual increase, whereas, as a matter of fact, there is probably a decrease in the amount of secretion. The apparent increase is due to the greater viscosity of the secretion, which renders it less absorbable by the ingoing current of air.

If the inflammatory process continues, a true hypertrophy of the tisues takes place on account of the increased nutrition from the large blood supply.

It is because of the different types of pathology associated with the inferior conchæ that there are so many types of treatment.

Where there is a simple functional disturbance, topical applications of various kinds will usually suffice. Those of a more chronic nature will often respond to electrical or chemical cauterization, or incising the turgescent bodies.

It is with the true hypertrophic types that we have a variation of opinion.

As can be seen from the pathology, local treatment can never hope to restore the nose to normal. However, if carried out persistently it will give great relief.

In the treatment of hyperplasias the object of most procedures is to remove the excess bone and tissue; often there is insufficient consideration of consequences.

It has been generally acknowledged that destruction of too much of the mucous membrane by cautery, or by cutting, leaves an area of scar tissue which is more detrimental than the original condition.

The solution of the problem, we believe, is the submucous resection of the turbinate.

Too much care cannot be taken in cleaning the nasal cavities previous to operation by the use of nasal sprays, douches, applications and instillations. Attention should be given to the general health of the patient.

The operative procedure is as follows: The turbinate to be resected is carefully cleansed of all incrustations. Then under local anesthesia I make an elliptical incision (in contradistinction to that of Odeneal) over the most prominent area of the inferior turbinate and resect this portion of the mucous membrane. My reasons for this step are as follows: The excess of mucous membrane is at once removed; the hypertrophied bone is more accessible, due to the fact that it is difficult to retract in the nose the large flap overhanging the area to be cut. The remainder of the mucous membrane and submucous tissue is easier to separate because of the accessibility. As much of the bone is then excised under direct vision as is necessary.

All bleeding is checked. The two flaps are allowed to fall into apposition and two sutures are taken to keep the margins in apposition so that when the tampon, saturated with bismuth and castor oil, is inserted it will not displace the flaps and defeat the purpose of this operation.

I have done three operations using the above technic. There was primary union in all three cases. To date none of them show any of the undesirable effects of previous procedures. There is ample breathing space, the unhealthy tissue has been removed, the rhinitis with turgescence has been relieved, and there is clear drainage from the sinuses.

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XXXII.

THE TREATMENT OF LARYNGEAL DIPHTHERIA: A STATISTICAL STUDY OF 655 CASES.

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WICHITA FALLS, TEXAS.

The following is an analysis of 655 successive cases of laryngeal diphtheria that have been observed and treated at Isolation Hospital, St. Louis, Mo., from April 1, 1924, to April 1, 1935.

All of these cases had either (1) laryngeal obstruction with a membrane on the tonsils, in the nose or in the larynx; or (2) laryngeal obstruction with a positive culture for diphtheria, and in either case were diagnosed as cases of laryngeal diphtheria at the time of being in the hospital.

The treatment followed was instituted by Dr. Curtis H. Lohr in 1924, continued by Dr. B. I. Krehbiel, and then myself, all successive in the position of Superintendent and Chief Resident Physician at Isolation Hospital.

In making a statistical study of these 655 cases of laryngeal diphtheria, the charts presented in this paper include all of the cases.

Laryngeal diphtheria may be classified into two types: primary and secondary. Primary laryngeal diphtheria is called such because the disease apparently started in the larynx without any evidence of the disease (membrane) in the nose or throat. These comprised 91 cases, or 13.9 per cent. Secondary laryngeal diphtheria included those cases with a membrane present in the nose or throat, or both.

Certain factors were found to influence the incidence of the disease.

1. Age plays an important part, shown by Chart 1.

From this chart, which shows the incidence, according to the age of the patient in years, it can be seen that most of the cases were from one to nine years of age; 84 per cent were under seven years of age and 95 per cent were under nine years of age.

2. Seasonal variation in the incidence was quite marked, shown by the following Chart 2 of the percentage of cases admitted during each month over the eleven-year period.

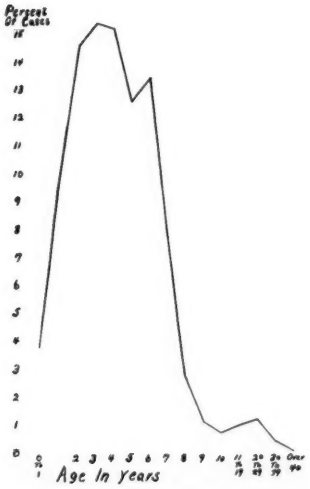

Chart 1. Incidence according to age.

From Chart 2 it can be seen that the peak number of cases occur during the months of February, March and April, and that very few cases occur during the months of August and September.

3. Yearly variation in the number of cases was plotted in Chart 3. (The fiscal year at Isolation Hospital starts April 1.)

4. Social status seems to be an important factor in so much as most of our cases came from homes of poor hygienic conditions, with which go ignorance, malnutrition and neglect;

5. Previous immunization against diphtheria had been performed in practically none of the patients:

6. Duration of illness before medical care is sought is a factor in the incidence of laryngeal diphtheria, because other forms of diphtheria can be aborted in their progress before involving the larynx. In our series of cases 79½ per cent were known to have been sick at least three or more days, and 29 per cent had been sick seven or more days before medical care was sought;

7. Tonsils were present in 98½ per cent of the patients, and though probably not of as much significance as the statistics would indicate, because tonsils are not usually removed in the age group of this series of patients, nevertheless they may be a fertile field for the growth of diphtheria bacilli;

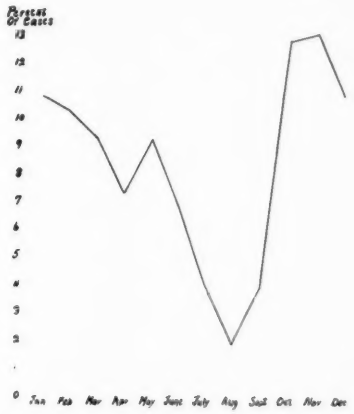

Chart 2. Incidence according to months.

8. Blondes seemed to be in greater preponderance, though we have no accurate figures to substantiate our opinion;

9. Race and sex seemed to play no part in the incidence of laryngeal diphtheria.

The important points in the treatment of laryngeal diphtheria are antitoxin, nursing care and medical care.

The nursing care of laryngeal diphtheria patients is very important. While the patient is in the acute stage of the disease, with respiratory obstruction, there should be a nurse in constant attendance. This nurse should be particularly trained to know what symptoms and signs are indicative of increasing obstruction and their relative importance so as to know when to call the doctor. She should be acquainted with the tricks of forcing fluids and quieting a child who is restless and apprehensive of respiratory embarrassment. Lastly, she should keep an accurate record of the medications given and of frequent regular observations of the pulse and respiration.

The antitoxin was given in a single dose of 20,000 units immediately upon entrance. This was all given intramuscularly unless the case was having much obstruction to respiration; then 2,000 units of this was given intravenously. Cases of primary laryngeal diphtheria that appeared to be cases of spasmodic croup were given 10,000 units of antitoxin on entrance and then later, when a positive culture was returned or on examination a membrane was found in the larynx, making the diagnosis laryngeal diphtheria, an additional 10,000 units of antitoxin was given.

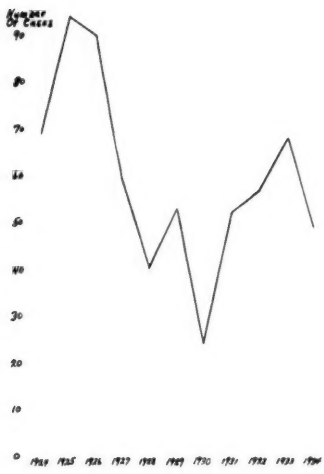

Chart 3. Incidence according to fiscal years.

The medical care must be under the supervision of well trained physicians, experienced particularly with this disease, and who are available at any time of the day or night.

After giving antitoxin the medical care consisted in the use of a steam tent with tincture of benzoin, using two drams to the quart of water; antispasmodics, such as antipyrin and sodium bromide; expectorants, such as potassium iodide; and sedatives, such as Dover's powders. A child, six years of age, was given ten grains of sodium bromide, two grains of antipyrin, and three minims of saturated solution of potassium iodide every four hours, and two grains of Dover's powders as necessary for restlessness. These medications were continued until improvement, when they were gradually discontinued.

Those patients who did not get along satisfactorily with the above medical treatment, inasmuch as the respiratory obstruction was not relieved, were given mechanical relief. The indications that we were governed by in feeling that relief of obstruction was needed, were increasing depth of the retractions of the intercostal spaces and sternum, increasing pulse rate to above 160 per minute, pulsus paradoxicus, decreasing respiratory rate, increased cyanosis, restlessness, and finally fatigue. Those who needed mechanical relief of obstruction were first given a trial with the intubation tube. I believe that the first intubation or tracheotomy done on a patient should be held

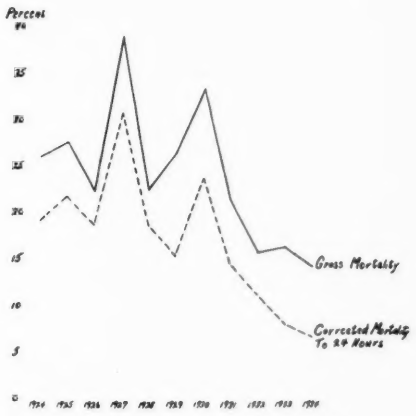

Chart 4. Mortality according to fiscal years.

off as long as possible unless endangering the life of the patient. The first intubation tube was always left in seventy-two hours before removal. If the tube was coughed up during that time, it was reintroduced if necessary. If the patient could not get along without the tube after seventy-two hours of intubation, he was reintubated for forty-eight more hours. In cases that coughed the tube up and cases in which the tube had to be removed before the above time was up, because of obstruction of the tube, the patient was never intubated more than five times.

Intubation is just like many other surgical procedures. For perfection, one needs some skill and considerable practice. In Isolation Hospital we had the patient held upright in the lap of a nurse and another nurse held the child's head. The important thing, we felt, was to have the head and body in a straight line with the chin out slightly. The mouth is held open with a mouth gag, preferably the Whitehead type. With one index finger the space between the two arytenoids and the epiglottis is located. The tip of the intubation tube is inserted in this space, loosened from its carrier, and pushed down with the index finger, to be covered over with the epiglottis.

Extubation may be done by several different methods. We first attempt to remove the tube by bobbing. The patient, wrapped in a mummy sheet, is held on a table with its head over the edge, face down, and pressure is exerted on the trachea, milking upward as the head is flexed, and the tube will fall on the floor. This was successful in the majority of cases. The other methods consist in removing the

Chart 5. Mortality of all cases and incidence of bronchopneumonia according to fiscal years.

tube with instruments, either directed entirely by touch, using the extubator, or under direct vision through the laryngoscope. The latter two methods are much more difficult.

On analysis of all the cases that required mechanical relief of obstruction by intubation or tracheotomy or a combination of both, which totaled 406 cases, 44, or 10.8 per cent, were so acutely obstructed as to require immediate tracheotomy. Of the remaining 362 cases on whom intubation was first tried, 204, or 50.2 per cent of the cases, could get along without the intubation tube after it had been left in for a total of less than six days. We felt, therefore, that those cases that require mechanical relief with the intubation tube for a period longer than a total of five days should have an elective tracheotomy because those cases will probably need mechanical relief much longer. From examination of the larynges with the laryngoscope it was found that those cases that need mechanical relief longer than a total of five days usually have secondary infection of the larynx with other organisms than the diphtheria bacillus. These secondary invading organisms, such as the streptococcus, staphylococcus and pneumococcus, tend to cause much more scar tissue formation than the diphtheria bacillus, resulting in a cicatricial stenosis of the larynx. This is particularly true when there is a chronic irritation associated with the infection, such as that of an intubation tube in the larynx.

In most cases a direct laryngoscopy was done. Where possible, pieces of membrane were aspirated or picked out of the larynx to

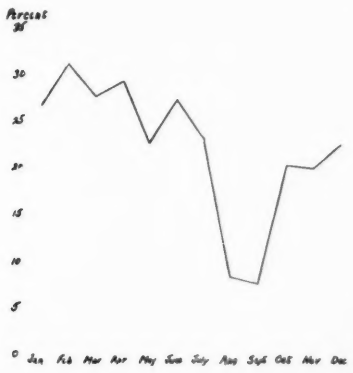

Chart 6. Mortality according to months.

give more air space. In most cases where there was considerable secondary infection, evidenced by marked redness, edema and induration of the structures in the larynx, we were more prone to do a tracheotomy.

After deciding to do a tracheotomy on a patient, an elective tracheotomy was done whenever possible, rather than to wait and have to do an emergency tracheotomy with added strain and risk to the patient. By an elective tracheotomy is meant one done in the operating room, under local anesthesia, with the intubation tube in place, using sterile technic and absolute hemostasis, removing the intubation tube after the tracheotomy has been completed.

The important point in doing a tracheotomy, I believe, is in having the patient in a position so that the trachea is prominent. This is easily accomplished by placing a sand bag under the shoulders. The anesthetic of choice is local anesthesia. The incision should be long, extending from the prominence of the thyroid cartilage to the suprasternal notch. After cutting through the skin, blunt dissection should be used to avoid cutting the numerous blood vessels that are forever presenting themselves. All bleeders should be tied. The trachea should be incised anywhere from the second to the fifth ring, being sure not to cut the first, or permanent damage to the cricoid may result. The wound is not sutured because it is essentially a contaminated wound and should have free drainage.

As to the time the tracheotomy tube should be left in, the case should be individualized. Unless contraindicated by some other condition, such as myocarditis, a laryngoscopy was done as soon as the

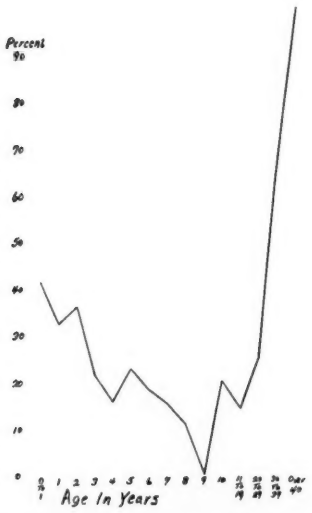

Chart 7. Mortality according to age.

acuteness of the disease was over, to determine the conditions existing in the larynx, particularly in reference to air space. If there was just a thin web of scar tissue occluding the space, this was dilated. If the occlusion was due to induration of the larynx with scar tissue, this was gradually dilated at weekly intervals. Suffice it to say that the treatment of laryngeal stenosis due to scar tissue is quite a problem, and fortunately, during the last several years, with the treatment outlined above, we had very few such cases to take care of. When there appeared to be sufficient air space in the larynx, decannulation was started by using increasing sizes of rubber corks in the tracheotomy tube until the child could breathe satisfactorily with the tube completely stopped. After having the tube completely closed for several days, it was then removed.

The treatment of the complications of laryngeal diphtheria is important in preventing fatalities in the more severe cases. Dehydration is often very severe in the patients on entrance because the parents were not insistent on the child taking fluids. It is important that the fluid balance be resumed in order to reduce toxicity. This we have done by the patient forcing of fluids by mouth, using the ordinary nursing bottle, even on the older children, and in infants sometimes fluids by mouth must be administered with a medicine dropper. Glucose, as a 5 or 10 per cent solution intravenously, or Hartmann's solution subcutaneously was definitely of value in combating acidosis.

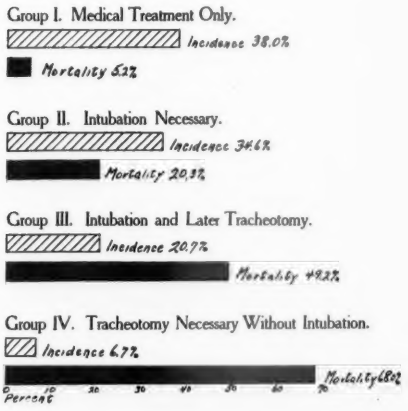

Chart 8. Incidence and mortality according to surgery necessary.

Acute toxic myocarditis makes its appearance usually seven to fourteen days after the onset of diphtheria. It was treated with absolute bed rest, seeing that the patient does not exert himself for anything, and the use of strychnin in large doses, such as one-fortieth of a grain every four hours, day and night. This treatment was continued at least six weeks, and longer when necessary, until complete recovery. Digitalis was not used because it was felt that digitalis acts synergistically with the diphtheria toxin to produce a heart block. The cases with toxic peripheral neuritis, including paralysis of the soft palate, absent knee reflexes and other isolated paralyses were treated the same as those having clinical signs of toxic myocarditis, because the hearts in those cases probably have absorbed just as much toxin as the peripheral nerve endings, and probably there exists a subclinical toxic myocarditis. Cases of paralysis of the palate or loss of pharyngeal reflexes were fed and given fluids with a nasal tube in order to prevent an aspiration pneumonia.

Bronchopneumonia was treated by the use of expectorants, such as potassium iodide and ammonium chloride. In those cases in which there was a progression of the involvement, inhalations of carbon dioxide, 10 per cent, in oxygen were given ten minutes every hour, and in those cases in which there was marked cyanosis, oxygen was administered by nasal catheter.

Other complications, such as serum sickness, pyelitis, otitis media and sinusitis were treated symptomatically.

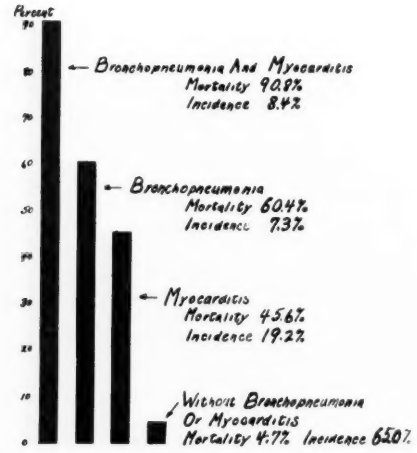

Chart 9. Mortality of cases of bronchopneumonia, myocarditis, and uncomplicated cases.

The mortality rate of laryngeal diphtheria seems to be affected by a number of factors:

1. Yearly variation seems to be quite a factor, shown by Chart 4.

A "corrected mortality rate to twenty-four hours" is a mortality rate of only the cases that do not die in the first tweny-four hours of hospitalization. We consider such a "corrected mortality rate to twenty-four hours" because in a public institution, such as Isolation Hospital, many moribund patients are received, and it is felt that those cases that die in the first twenty-four hours of hospitalization should not be counted in a mortality rate reflecting on treatment. We would like to believe that the above reduction in mortality rate was in part due to improved treatment.

2. The incidence of serious complications, we believe, has an effect on the mortality rate shown by Chart 5, which I think explains the high peak of 38.9 per cent mortality rate in 1927 as being partly due to the high incidence of bronchopneumonia that year, 30.5 per cent of the cases having bronchopneumonia.

The comparatively high mortality rate of 33.3 per cent in the year of 1930 should not be considered seriously in reference to the mortality rates of the other years because of the small number of cases that year (refer to Chart 3—"Incidence According to Fiscal

Years"). There were only twenty-four cases during the fiscal year 1930 compared to an average of sixty-three cases during the other ten years.

3. Seasonal variation as a factor is shown by Chart 6, exhibiting a low mortality rate of around 8 per cent during the months of August and September, compared to a mortality rate of over 20 per cent for the rest of the months of the year.

4. Age seems to play an important part in the mortality rate shown by Chart 7.

The curves for the mortality rates over eleven years of age are not accurate mathematically, because of the small number of cases over eleven years of age shown in Chart 1 earlier in this paper.

5. The surgery necessary seems to definitely affect the mortality rate when the above rules for intubation and tracheotomy are followed, not meaning that the surgery done makes the prognosis worse, but that those cases that require more mechanical relief have a much poorer prognosis, as shown by Chart 8, in which the cases are divided into four groups according to the manner in which relief of obstruction to respiration was obtained.

6. The complications present definitely affect the mortality rate shown by Chart 9, in which the cases which had bronchopneumonia and myocarditis; those which had only bronchopneumonia; those which had only myocarditis; and those which had neither of those complications have been separated from each other and their mortality rates charted.

SUMMARY.

This paper, a statistical study of 655 cases of laryngeal diphtheria observed at Isolation Hospital during the past eleven years, shows certain factors influence the incidence of disease and others influence the mortality, while the above line of therapy was followed.

The incidence is apparently affected by age, season, yearly variation, social status, previous immunization against diphtheria, duration of illness before medical aid is sought, and the presence of tonsils.

The mortality rate is apparently affected by yearly variation, seasonal variation, age, necessary surgery, and complications present.

1300 S. EIGHTH STREET.

XXXIII.

OTOSCLEROSIS

INDEX OF THE LITERATURE ON OTOSCLEROSIS FROM 1928-1935

Issued by the Central Bureau of Research

of

THE AMERICAN OTOLOGICAL SOCIETY, INC.

The following index constitutes Section 2 of the new Volume III on Otosclerosis. This volume will be issued shortly by the Central Bureau of Research of The American Otological Society, Inc., and is published here by special arrangement with the ANNALS. The first portion of this volume, consisting of an abstract of the literature, was published in the March (1936) issue of the ANNALS, Volume 45, No. 1. The voluminous literature is not completely covered in the text, but no references to otosclerosis have been knowingly omitted from the Annotated Bibliography. The table of subjects is given for reference from the Annotated Bibliography.

EDMUND P. FOWLER.

OTOSCLEROSIS

VOLUME III

(Continued)

INDEX OF THE LITERATURE ON OTOSCLEROSIS, 1928 TO 1935, INC.

1. Authors' Index, according to subjects (numbered as in Contents).
2. Annotated Bibliography:

   The figures in brackets refer to the subjects under which the author is quoted.

   Annotations follow the title, medium, page and date of publication.

   Because of the possible future application of several references not directly related to otosclerosis, it was thought advisable to include them in the Annotated Bibliography.

SUBJECTS.*

VOLUME III.

1. Heredity
2. Embryology
3. Regression
4. Bone Pathology
5. Circulatory and Inflammatory Factors. Venous Stasis
6. Vasomotor Mechanism
7. Nervous Mechanisms (Cortical, Central, Nerve and End Organ)
8. Endocrinology
9. Metabolism
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11. Animal Experimentation
12. Psychology
13. Diagnosis. Prognosis. New Tests
14. Treatment, Surgical
15. Treatment, Non-Operative

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*Numerical references in parentheses in the Bibliography refer to the sections in this table. See also correspondingly numbered chapters, page 171 et seq. ANNALS, March, 1936.

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BAUER, J., and STEIN, C.: Die Bedeutung der Konstitution in der Pathogenese der Otosklerose. *Zeitschr. f. angew. Anatomie*, 1:546, 1913-14.

The significance of the constitution in the pathogenesis of otosclerosis. These investigations (26 cases) indicate that similarly as in hereditary-degenerative deafness, also in otosclerosis the manifestations of a general transmissible constitutional anomaly are demonstrable. (9)

—— Vererbung und Konstitution bei Ohrenkrankheiten. *Zeitschr. f. d. ges. Anatomie, Abt. 2*, 18:483, 1924-5.

Heredity and constitution in ear diseases. Contributions to clinical-constitutional pathology. Abnormal heredities originate through so-called germ variations or idiokinesis, i.e., through alteration of one or several genes under the influence of some action on the parental organism. (1, 9)

—— Blutdrüsenerkrankungen u. Gehörorgan. (Diseases of the endocrinic glands and the auditory organ.) *Alexander- Marburg. Handbuch der Neurologie des Obres.* 3:787, 1926.

There exists no endocrinic gland whose operative removal or implantation or whose hormone when introduced into the organism would give rise, constantly or even regularly, to symptoms of lost function or of irritation, or to anatomical changes, respectively, in the domain of the auditory organ. (8)

BAYLISS, SIR W. M.: Vasomotor System. *Monograph. London, Longmans, Greene and Co.*, 1923. 163 pp.

BECK, J.: Zur Klinik und Roentgenologie der Ostitis deformans (Paget) der Innenohrkapsel. *Zeitschr. f. Hals-, Nasen u. Ohrenhlk.*, 35:17, 1933.

Incidence and roentgenology of ostitis deformans (Paget) of the inner ear capsule: Roentgen picture of the left petrous bone in ostitis deformans, showing faint and indistinct designs of the labyrinth capsule. (4)

BECKS, H., and WEBER, M.: Influence of diet on bone system with special reference to alveolar process and labyrinthine capsule. *J. Amer. Dental Assoc.*, 18:197, 1931.

BEHRENDT, H., and BERBERICH, J.: Zur Stoffwechselpathologie der Rachitis und Otosklerose. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 121:280, 1929.

Pathological metabolism in rachitis and otosclerosis: Intermediate disturbances exist in otosclerosis which in many ways are analogous with those of rickets. The metabolic equilibrium differs from the conditions in

normal persons and more particularly the balance between acid and alkaline valences appears to be regularly altered. (9)

—— Stoffwechseluntersuchungen bei Otosklerose. *Zeitschr. f. d. ges. exper. Med.*, 78:310, 1931.

Metabolic changes in otosclerosis: These metabolic investigations led to the conclusion that in otosclerosis the regulation of the acid base economy deviates constitutionally toward the acidosis side. The basal metabolism is lowered, or at the lowest boundary of the normal standard. Eight tabulations. (9)

BENESI, V. O.: Linksseitige Otosklerose. *Monatsschr. f. Ohrenhlk.*, 66:877, 1932.

Left-sided otosclerosis. Demonstration of specimens.

BERCERUELO, S. R.: La Otospongiosis y su calcemia. *Revista Espan. y Amer. de Laringol. Otol. y Rinol.*, 24:24:110, 1933.

Otosclerosis and its blood calcium contents: This is a hypocalcemic hereditary disease, affecting especially women. 64 observations. Hypercalcemia was present in 20 per cent of the cases, always in neurotic patients. The hypocalcemia is not apparently related to the degree of functional deficiency of the ear or the severity of the tinnitus. Bibliography. 84 references. (9)

BERGGREN, S.: Ein Versuch operativer Behandlung von einem Fall hochgradiger Otosklerose. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 122:24, 1929.

An attempt at operative treatment of a case of advanced otosclerosis. (14)

BERNARD-LAPOMMERAY: Otospongiose et hérédosyphilis. *Rev. de laryngol., otol., rhinol.*, 54:523, 1933.

Otosclerosis and congenital syphilis: Report of a case of otosclerosis developing on the basis of congenital syphilis. (13)

BERRY, GORDON.: The psychology of progressive deafness. *Journ. Amer. Med. Assoc.*, 101:1599, 1933.

Nine commandments for the hard of hearing. Certain rules of conduct applicable to almost every deafened person who seeks an otologist. (12)

BIRKHOLZ.: Zur medikamentösen Behandlung der Otosklerose. *Monatsschr. f. Ohrenhlk.*, 64:558, 1930.

Medical treatment of otosclerosis: Courses of intravenous calcium bromide therapy, systematically applied, exert a favorable effect on moderately grave cases of otosclerosis. (15)

—— Zur medikamentösen Behandlung der Otosklerose. *Monatsschr. f. Ohrenhlk.*, 68:1023, 1934.

Medicinal treatment of otosclerosis: Experimentation with vigantol gave negative result. Better results from intravenous supply of calcium bromatum, in over a dozen cases. (15)

BOBONE, T.: Ancora di una cura dell' otosclerosi. *Boll. d.Mal. Orecchio*, 52:549, 1934.

Another treatment for otosclerosis: Favorable results from adrenalin medication with repeated progressive injections. (15)

BONAIN, A.: Étude sur les fenêtres labyrinthiques et leur rôle fonctionnel. *Arch. internat. de laryngol. otol. rhinol.*, 9:783, 1930.

A study of the labyrinth windows and their functional role. The phylogenesis of the apparatus of equilibrium and audition in the various animal

species indicates that the oval window, the tympanic membrane and the chain of ossicles are solely designed for the protection of the labyrinthine organs, as well as for auditory accommodation. The round window, the most direct and most permeable route for access of the sound waves to the sensory auditory elements, has the function of transmitting the sounds to the internal ear. (2)

BOQUET, R.: Contribution a l'étude du syndrome de Spurwayvan der Hoeve: *sclérotiques bleues, fragilité osseuse congénitale, surdité. Paris Thesis*, 1932.

Contribution to the study of the syndrome of blue sclerotics, congenital bony fragility, deafness.

BORDIER, H.: Nouvelle méthode électrothérapique dans les bourdonnements. *Arch. electr. méd.*, 43:37, 1935.

New electrotherapeutic method for aural tinnitus and hardness of hearing.

BORGHESAN, E.: Paratiroidi e orecchio interno. *Valsalva*, 6:213, 1930.

Parathyroids and internal ear. Italian text. Anatomo-pathological study in experimental parathyroid insufficiency: In rabbits, changes of the labyrinth capsule are elicited comparable to those of the first and the beginning of the second stage of otosclerosis. 6 Figs. (11)

BOZZI, E.: Osservazioni sull'origine e sul decorso dell' arteria uditiva interna. *Otologia Italiana*, 4:464, 1934.

Observations on the origin and course of the internal auditory artery, based upon personal investigations conducted on 105 male and female cadavers. (6)

BREITMANN, M.: Ueber die endokrinen Grundlagen der Otosklerose. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 136:117, 1933.

The endocrinic bases of otosclerosis. Correct endocrinic diagnosis and treatment can only be entirely individual. In each case, first the endocrinic formula, then the sequence of affections of the various glands, must be determined. (8; 9)

—— Ueber die endokrinologische Diagnostik in der Ohren- Hals- u. Nasenheilkunde. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 135:289, 1933.

Endocrinological diagnosis in otorhinolaryngology. Systematic endocrinological examination of different otosclerosis cases showed different endocrine affections. An endeavor must be made to discover and treat the primarily affected gland. (8)

BRICKNER, R.: Mental effects of acquired deafness. *Conference*, 1930.

A careful review of the literature showed that the mental effects of acquired deafness represent a subject practically untouched by psychiatrists. (12)

BRUHL, G.: Bemerkungen zu O. Mayer's Arbeit "Ueber die Entstehung der Spontanfrakturen der Labyrinthkapsel und ihre Bedeutung für die Otosklerose." *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 26:587, 1930.

Comments on O. Mayer's article on the origin of spontaneous fractures of the labyrinth capsule and their significance for otosclerosis, which in Brühl's opinion is a constitutionally caused, degenerative disease. The mechanical relations in the temporal bone were first stated by him in explanation of the focal behavior and bony changes, especially in the vestibular window niche. (2; 4)

—— Zur Knöchernen Stapesankylose. *Passow-Schaefer's Beitr. z. Anat. Physiol. Pathol. u. Therap. des Ohres*, 4:71, 1910.

Contribution on osseous ankylosis of the stapes. (10)

—— Otosklerose. *Ergebn. d. ges. Med.*, 1:376, 1920. *Deutsche. med. Wchnschr.*, 46:944, 1920.

Otological advice for the practioner. The prognosis of otosclerosis is not favorable, but the process often remains stationary and does not regularly terminate in deafness. Otosclerosis is incurable and therapy must be restricted to the relief of symptoms and the protection of the internal ear. (10)

BRUNO, D.: Il Nucleosyl nella cura della sordità progressiva da otosclerosi. *Giornale di Oculistica*, 11:115, 1930.

Nucleosyl in the treatment of progressive otosclerotic deafness. (Double nucleinate of manganese and calcium.)

BRUNNER, H.: Beitrag zur Histogenese des otosklerotischen Knochens. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 6:320, 1923.

Contribution to the histogenesis of otosclerotic bone. (9)

—— Ueber die Veränderungen des Schläfenbeins bei Ochronose. *Monatsschr. f. Ohrenhlk.*, 63:997, 1929.

Changes of the temporal bone in ochronosis. (9)

—— Zur Pathologie der Ostitis deformans (Paget) des Schläfenbeines. *Klin. Wchnschr.*, 10:2174, 1931.

Contribution to the Pathology of ostitis deformans (Paget) of the temporal bone. Ostitis deformans represents a chronic inflammatory process in acute installments. In Paget's disease of the temporal bone, nodular infiltrations are locally found in the marrow spaces; otitis interna may follow, proving the inflammatory nature of ostitis deformans. (4)

—— Ueber Spaltbildung in der knöchernen Innenohrkapsel. *Wiener. klin. Wchnschr*,. 44:173, 1931.

Gap-formation in the bony inner ear capsule; Based on examination of serial sections of twenty-six petrous bones with various pathological changes in the middle and internal ear. Gaps in the semicircular canal region were seen in twenty-four petrous bones. (4)

—— Ein Fall von Ostitis fibrosa (Paget) des Schläfenbeines. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 30:110, 1931.

A case of ostitis fibrosa (Paget) of the temporal bone. Microscopical findings in the temporal bones of a man sixty-five years of age. Illustrated. 7 Figs. (4)

BRYANT, B. L.: Hypothyroidismus in der Otolaryngologie. *Monatsschr. f. Ohrenhlk.*, 66:387, 1932.

Hypothryoidism in otolaryngology. Contribution to the Hajek Festschrift. German text; translated from English by Waldapfel. (15; 8)

BRYANT, W. SOHIER.: Pathology and prognosis of chronic progressive hardness of hearing: otosclerosis. *Ann. of Otol., Rhinol. and Laryngol.*, 17:652, 1908.

"Certain areas of the tympanum, the oval window, promontory and drum membrane, are most often affected, because these areas are more subject to reflex vasomotor and other vascular changes.—We know that the tympanum is subject to vasomotor changes, which finally cause diminution of blood supply and atrophic changes." (6)

BUNCH, C. C.: Some observations on clinical otosclerosis. *Ann. of Otol., Rhinol. and Laryngol.*, 43:344, 1934.

This contribution is not presented as a completed study. It contains numerous quotations from the literature collected in the first volume of "Otosclerosis." (13)

CABOCHE, H., and OUMIKOVA: Quelques cas d'otopathies traités par applications de haute fréquence. *Arch. internat. de. laryngol., otol. rhinol.*, 35:416, 1929.

Some cases of ear disease treated by high frequency applications. (15)

CALDERIN, A. M.: Sordera capsular y endocrinologia. *Medicina Ibera*, 2:491, 1933.

Capsular deafness and endocrinology.

CALICETI, V.: Le sindromi vertiginose del labirinto. *Arch. ital. otol.*, 45:65, 1933.

The vertiginous syndromes of the labyrinth: Report of two cases on the basis of which the various pathogenetic theories are discussed, which have been propounded for the elucidation of this syndrome. (6)

CANTELE, P. G., and GRAHE, H.: Der Einfluss des Sympathikus auf die vestibuläre Erregbarkeit. *Arch. f. Ohrenhlk.*, 130:281, 1932.

Influence of the sympathetic nerve upon vestibular irritability: These investigations show, in rabbits, an influence of sympathetic nerve stimulation and paralysis upon the vestibular irritability. The findings contraindicate a vasomotor explanation of caloric nystagmus. (11; 6)

CANTELE, P. G.: Sistema labirintico e reflessi vasomotori e respiratori; ricerche sperimentali. *Arch. ital. di otol.*, 44:129, 1933.

Experimental research on the labyrinthine system and the vasomotor and respiratory reflexes. Extensive survey of previous contributions. Experiments on rabbits. Destruction of the peripheral labyrinth is followed by a few irregular fluctuations of the blood pressure, which finally drops slowly and permanently. (6)

CARCO, P.: Sulla presclerosi timpanica di Citelli. *Communication, 2 Congrés internat. d'otorhinolaryngol., Madrid*, 1932. p. 99.

Citelli's tympanic presclerosis: Report of five cases confirming the existence of the presclerosis discovered by Citelli, and showing the excellent results which during this phase can be obtained by iodine treatment. No calcium medication was added in these cases, but very favorable results were accomplished. Also Citelli in his cases employed iodine treatment alone. (15)

—— Sulla presclerosis timpanica di Citelli. *Arch. ital. di otol., rhinol. e laringol.*, 46:545, 1934.

Notes on Citelli's presclerosis with report of five new cases. The treatment must consist chiefly in surprising the disease in the phase of presclerosis, when excellent results are possible through iodine - and calcium therapy. (15)

CATHCART, G.: Chronic deafness. *Proc. Roy. Soc. Med.*, 22:1421, 1929.

Discussion on treatment of deafness. A total of 623 cases of chronic deafness, including 172 cases of otosclerosis, were treated by the electrophonoid method alone; 90 of the otosclerosis group were improved and 82 showed too slight improvement to warrant continuance. (15)

—— The alleviation of chronic deafness. *Practitioner*, 124:142, 1930.

CHAJUTIN, I. M.: Zur Frage der Aetiologie und Therapie der Otosklerose. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 127:61, 1930.

Problem of the etiology and therapy of otosclerosis. Interpretation of otosclerosis, with its displacement of the calcium ions equilibrium, as a

pluriglandular affection. An important part is apparently played by parathyroid and ovarian disturbances. (9)

—— Aetiologie und Therapie der Otosklerose. *Zentralbl. f. Hals- Nasen- u. Ohrenblk.*, 19:128, 1932.

Etiology and therapy of otosclerosis. By the Russian author, otosclerosis is regarded, not as a local, but as a general disease. Treatment of otosclerosis should consist in a direct or indirect action upon the endocrinic apparatus. (9)

—— Troubles ovariens et otosclérose. *Gynéc. et obst.*, 27:146, 1933.

Ovarian disturbances and otosclerosis: Report of three cases of otosclerosis in women with menstrual disturbances apparently due to ovarian dysfunction, relieved by subcutaneous implantation of bone. The otosclerosis was not benefited by the rise in blood calcium. It is presumably associated with a pluriglandular dysfunction, not ovarian dysfunction alone. (8)

CHARSCHAK, E.: Die Otosklerose nach dem Material des Internationalen Otolaryngologischen Kongresses zu Madrid, September, 1932. *Acta Otolaryngol.*, 19:352, 1934.

Otosclerosis according to the material of the International Otolaryngological Congress in Madrid, September, 1932. Brief compilation of the statements rendered on the subject of otosclerosis at the Congress. Views on etiology and course of the otosclerotic bone process are still widely divergent.

CHIARINO, A., and ALONSO, J. M.: Otra suerte de tratamiento quirurgico en la otosclerosis. *Anales Otologia*, Uruguay, 5:81, 1935.

New mode of surgical treatment of otosclerosis: Ligation of the inferior thyroid artery, to modify the parathyroid function. Claim of beneficial results in three cases. (14)

CIGNA, V. de.: Un nouveau phénomène acoustique et une nouvelle méthode d' acoumétrie. *Ann. d. mal. de l'oreille*, 49:501, 1930.

A new acoustic phenomenon and a new acoumetric method. Communication to the 24th Congress of the Italian Otorhinolaryngological Society, Catania, 1928. Preliminary note. The pneumotympanic test furnishes a useful method of examination of the auditory function. (13)

CITELLI, S.: Contributo alla conoscenza della cosidetta otosclerosi. *Arch. ital. di otol.* 25:29, 1914. *Arch. internat. de laryngol.* 36:681, 1913.

Contribution to the knowledge of so-called otosclerosis: This affection can be produced by the most variegated diseases, among which probably predominate the diseases of the endocrinic glands, especially the hypophyseal system, and general intoxication of uremic origin. Rather than as a single pathological unit, otosclerosis should be considered as a syndrome to be designated as the otosclerosis syndrome. (8)

COATES, G. M.: Discussion of some of the recent developments in regard to otosclerosis, cholesteatoma and the static labyrinth. *Laryngoscope*, 43:622, 1933.

Definite otosclerotic areas, some of them apparently of long standing, may be found in the temporal bones of persons who have not shown the usually accepted symptoms of clinical otosclerosis during life.

CODY, C. C.: Relation of vitamins A, B, D and G to otolaryngology. *Arch. Otolaryngol.*, 16:661, 1932.

Dietetic deficiency in fat-soluble vitamins A and D induced changes of rachitic character in the bony labyrinth of rats, the effect of this deficiency corresponding to the changes in otosclerosis. But the osseous laby-

rinth of the rat in deficiency of vitamin A and D showed no change in hardness, calcium content, and also in auditory function. (9; 11)

COLONNA, P. C.: Osteogenesis imperfecta. *Amer. J. Surg.*, 15:336, 1932.

One of nine cases of osteogenesis imperfecta showed progressive deafness.

CORININ, Y.: Le renforcement ondulatoire. *Thèse de Paris*, 1931.

Undulating re-enforcement: The principal objective of undulatory re-enforcement is the localization of the site of the deafness. The test remains positive even for a very pronounced deafness when the lesion is purely labyrinthine. It is a much more sensitive test than the Rinne. (13)

COULET, G.: Note sur le rôle des autointoxications dans l'otite scléreuse. *Communication, 2 Congrès Internat. d'otorhinolaryngol., Madrid*, 1932. 149.

Note on the role of the autointoxications in otolsclerosis: Endogenic intoxications are undoubtedly among the most obscure causative factors of sclerosis of the auditory organ. They are produced through functional disturbances of the digestive tube and the liver as well as disturbances of the renal functions and are likewise due to changes of the antitoxic glands (suprarenals, thyroid), associated with deficient or excessive secretion.

GRINIS, M. de.: Anatomie der Hörrinde als Grundlage des physiologischen und pathologischen Geschehens der Gehörwahrnehmung. *Monograph, Berlin, J. Springer*, 1934.

Anatomy of the auditory cortex as the basis of the physiological and pathological incidence of auditory perception. (7)

CULLER, E.: Function of the round window. *Science*, 78:269, 1933.

The findings clearly support the "classical" theory of hearing in relation to the round window's function. Plugging the round window fossula in dogs reduces the animal's hearing and also the electrical pulses over the auditory nerve. (11)

CULLER, E., FINCH, G., and GIRDEN, E.: Function of the round window in hearing. *Amer. J. Physiol.*, 111:416, 1935.

CUNNINGHAM, H. K.: Some aspects of otosclerosis. *J. Tennessee Med. Association*. 27:306, 1934.

In the light of our present knowledge, otosclerosis is probably a local manifestation of a general systemic dysfunction. Heredity seems to have a certain definite bearing on this condition. (13)

CUSENZA, G.: Lesioni della capsula labirintica nell'acidosi sperimentale da somministrazione di HCL. *Valsalva*, 7:916, 1931.

Lesions of the labyrinth capsule in experimental acidosis through administration of hydrochloric acid. (11)

—— Timo e otospongiosi. *Valsalva*, 8:766, 1932.

Thymus and otosclerosis: Removal of the thymus gland, in experiments on hens, induced some areas of resorption in the labyrinth capsule, but not of the otosclerotic type. (11)

DAVENPORT, C. B.: Hearing in children when both parents have otosclerosis. *Trans. Amer. Otol. Soc.*, 20:48, 1930.

The high incidence of otosclerosis, in seven of eight cases in the children of two otosclerotics, suggests recessive inheritance. The bony defect upon which otosclerosis depends is apparently due to the absence of at least one genetic factor that makes for normal bone production and normal maintenance. (1)

DAVENPORT, C. B., MILLES, B. L., and FRINK, B. B.: The genetic factor in otosclerosis. *Arch. Otolaryngol.*, 17:135, 340; 502; 1933.

Family histories, tables and charts from original studies and from literature. Surmise of two dominant factors, one sex-linked, in the heredity of otosclerosis. 9 tables. (1; 13)

DAVENPORT, C. B.: The genetic factor in otosclerosis. *Amer. Med. Assoc., Chicago*, 1935.

DAVIS, J. E.: Different types of deafness. *Laryngoscope*, 40:20, 1930.

A study of the etiology, methods of diagnosis, prophylaxis and treatment. Specific glandular therapy, with other necessary treatment of metabolic disturbances, seem at present to be the most hopeful means of combating otosclerosis. (Bibliography, 25 references.) (13)

DAVIS, HALLOWELL.: The electrical phenomena of the cochlea and the auditory nerve. *Jrl. of the Acousical Society of America.* 6:205, 1935.

"The mechanical vibration causes the sensory cells of the organ of Corti to develop an electrical potential between their upper and lower ends. The energy for this electrical potential is derived from the sound waves. Whether this cochlear response is part of the mechanism for the excitation of nerve impulses or whether it has no physiological significance is still uncertain."

DELIENCOURT, R.: La diathermie dans le traitement des états de sclérose de l'oreille. *Prat. méd. franc.*, 11:344, 1930.

Diathermy in the treatment of sclerotic conditions of the ear. (15)

DESJARDINS, A. U.: Action of Roentgen Rays and radium on eye and ear. *Amer. J. Roentgenology*, 26:921, 1931. Sect. 3.

Review of the literature on animal experiments, analyses of experimental data, clinical consideration. Bibliography, 50 refrences. (15)

DELAMARE, G., and TANASESCO: Étude sur les artères du sympathique céphalique, cervical, thoracique et abdominal. *J. de l'anat. et de la physiol.*, 42:97, 1906.

Anatomical study of the arteries of the cephalic, cervical, thoracic and abdominal sympathetic nerve. (6)

DENIER: Ondes courtes et otosclérose. *Bull. Off. Soc. franc. d'électrotherapie et radiol.*, 41:386, 1932.

Short waves in otosclerosis: Report of four cases treated with short waves diathermy. Three patients showed definite improvement; one case, past 60 years of age, was not benefited. (15)

DESJARDINS, A. U.: Action of Roentgen rays and radium on the eye and ear. *Amer. Jrl. of Roentgenology*, 26:921. Sect. 3, 1931.

From the Mayo Clinic, Rochester, Minn. General considerations. Review of the literature on animal experimentation; analyses of experimental data; clinical considerations. Bibliography for ear, fifty references. (15)

DESPONS, J.: Otospongiose et maternité. *Bull. Soc. Med. de St. Luc*, 41:184, 1935. Otosclerosis and maternity.

DESSOFF, J.: Blue sclerotics, fragile bones and deafness. *Arch. of Ophth.*, 12:60, 1934.

A review of the various theories on the etiology of this disease. Emphasis is laid on the strong possibility of hyperparathyroidism as the causative factor.

DICKIE, I. K. M.: Chronic progressive deafness including otosclerosis and diseases of the internal ear. *Arch. of Otolaryngol.*, 19:712, 1934; 22:99, 1935.

Published under the heading of Progress in Otolaryngology, a summary of the bibliographic material available in the field of otolaryngology. Recent articles.

DILLON, J. G., and PREOBRASCHENSKY, B. S.: Ueber Pneumatisation des Schläfenbeins bei Otosklerose nach Ergebnissen der Roentgenographie. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 26:537, 1930.

Pneumatization of the temporal bone in otosclerosis, according to roentgenographic findings: This may vary as much in otosclerotics as in other persons. The structure of the temporal bone exerts no essential influence on the course of the otosclerosis. 37 references. (4)

DINOLT, R.: Osteopsathyrosis mit blauen Sklera. *Monatsschr. f. Ohrenhlk.*, 65:1015, 1931. *Austrian Otol. Soc.*, June 1, 1931.

Presentation of patient, woman of 35 years, with blue sclerotics, slowly progressive hardness of hearing since ten years. Very severe tinnitus, no vertiginous sensations. Middle ear deafness with greatly prolonged bone conduction. Stigmata suggestive of osteopsathyrosis. No signs of osteopsathyrosis in the family. (4)

DOEDERLEIN, W.: Histologische Untersuchungen über den Beginn der Otosklerotischen Knochenerkrankungen. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 22:293, 1928.

Histological investigations on the beginning of ostosclerotic bone affections. (4)

—— Otosklerose bei den Erkrankungen mit Hyperfunktion des lymphatischen Systems. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 134:24, 1933.

Otosclerosis in affections with hyperfunction of the lymphatic system: Based on examination of selected cases with incipient otosclerotic changes from about 200 petrous bones in the collection of the Berlin Ear, Nose, and Throat Clinic. (4)

DOWNING, J. A.: Otosclerosis. *J. Iowa Med. Soc.*, 18:421, 1928.

Review of some earlier investigations.

DRURY, DANA W.: Chronic deafness; an endocrine study of one thousand cases. *Laryngoscope*, 39:555, 1929. *New Engl. J. Med.*, 201:206, 1929.

Contribution from the Evans Memorial No. 193; A-103. Large series of cases studied intimately from the endocrine angle: thyroid, pituitary, ovaries. Non-endocrine group of 415 cases or 42 per cent. Otosclerosis is not an inflammatory condition. It is a degenerative process in the organ of hearing as a whole. (8)

DRY, T. J.: A case of osteogenesis imperfecta with familial history. *Proc. Staff Meet. Mayo Clinic*, 8:329, 1933.

Case report, woman with blue sclerotics, history of multiple fractures and deafness as the chief complaint. Positive family history in a grandparent, mother, collaterals, children and grandchildren. (4)

DUARTE, J.: Sobre la etiologia de la otospongiosi. *Med. ibera*, 1:73, 1929

The etiology of otosclerosis: Raises the question as to its causes being of endocrine origin. Spanish text.

DUERTO, J.: Modificaciones del aparato auditivo en la otospongiosis apreciable por otoscopia. *Med. ibera*, 1:589, 1928.

Modifications of the auditory apparatus in otosclerosis, demonstrable by otoscopy. (13)

—— Sobre un nuevo tratamiento de las oto-esclerosis. *Clin. y Lab.*, 15:222, 1930. *Revista Espan. y Amer. d. laringol.*, 21:241, 1930.

A new treatment of otosclerosis. Spanish text. Report on Sourdille's operation for the removal of otosclerotic symptoms. (14)

DUNDAS-GRANT, J., ORMEROD, F. G., and SIMMONDS, F.: Family with blue sclerotics, fragilitas ossium and otosclerosis. *J. Laryngol. and Otol.*, 44:273, 1929.

Presentation of such a family, at the Royal Society of Medicine, Section of Otology. The blue sclerotics increase in depth of color with every illness, in all the last two generations. In no case was there any fracture at birth. Blueness of sclerotics was found at birth and does not seem to have increased in depth. (4)

DUNDAS-GRANT, J.: Cases of aural vertigo amenable to treatment by ossiculectomy. *Lancet*, 2:1029, 1933.

Report of twelve cases. The indications for ossiculectomy are not very frequent, but often are very clear, and the reported results show they should be acted upon. (14)

—— Simplified method of determining the percentage of actual hearing power in tuning-fork tests. *J. Laryngol. and Otol.*, 49:233, 1934.

A simple physical experiment permits the determination of the time during which the vibration of the tuning fork is diminished by one half. By means of a fairly easy calculation the percentage is ascertained for the response of the patient as compared to the normal hearing of the examiner. (13)

ELLIS, R. W. B.: Four cases of fragilitas ossium and blue sclerotics. *Proc. Roy. Soc. Med.*, 24:1054, 1931.

The fourth case showed otosclerosis.

ESCAT, E.: Réalisation du tableau clinique de l'otospongiose par la périostite syphilitique. *Ann. d'otolaryngol.*, 1:203, 1931.

Simulation of the clinical picture of otosclerosis by syphilitic periostitis of the labyrinthine tympanic wall. Numerous investigations served to refute the surmise of otosclerotic changes being eventually referable to syphilis, acquired or congenital. Therapeutically, otosclerosis is not amenable to anti-syphilitic medication. (15)

ESCAT, E., and RIGAUD, P.: Methode de recherches a suivre pour résoudre le probléme de l'otospongiose. *Communication, 2 Congrès Internat. d'Otorhinolaryngol., Madrid*, 1932. p. 167.

Method of investigations to be followed, in order to solve the otosclerosis problem: To begin with, fundamental investigations should be, at least provisionally, restricted exclusively to typical otosclerosis, with its characteristic symptomatology. Otopathic associations (tubal catarrh, ear suppurations, labyrinth disturbances, etc.) should be noted and their eventual influence duly considered. Bony changes should be radiographically looked for, not only in the temporal bone, but also in the various other segments of the skeleton. (9)

ESCHWEILER: Ueber Spaltbildungen in der Labyrinthkapsel. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 34:152, 1933.

Gap formations in the labyrinth capsule: Description of the fissures found in specimens of the capsule, interpreted as artifacts due to the mode of preparation, not as fractures incurred during life. (4)

FALTA, L.: Otosclerosis and climacteric. *Zentralbl. f. Hals-, Nasen- u. Ohrenhlk.*, 15:549, 1930.

German abstract of Hungarian original. Observations comprise 129 patients, including 53 men and 76 women in whom otosclerosis appeared after the fortieth year of life; in eleven women during the climacteric; eight of these had borne several children without any impairment of hearing. Otosclerosis supervening in the climacteric is interpreted as a metabolic disturbance.

FAVRE, F.: Otospongiose et syphilis. *Communication, 2 Congrès International d'Otorhinolaryngologie, Madrid*, 1932. p. 128.

Otosclerosis and syphilis; Otosclerosis is not a direct manifestation of syphilis. It may sometimes be the manifestation of an endocrine disturbance of syphilitic origin, acquired or congenital. Syphilis plays only a restricted role and is often merely an intercurrent affection. All systematic specific therapy is objectionable. (15)

—— Faut-il admettre une otospongiose syphilitique? *Dissertation, Toulouse*, 1932.

Is a syphilitic otosclerosis admissible? Surmise that in a few exceptional cases, otosclerotic bone changes in the labyrinth capsule may be of syphilitic origin. Evidence of syphilis was found in at most 5 per cent. (13)

FEDERICI, F.: Nouvelle épreuve acoustique ayant surtout pour but de deceler une ankylose entre l'étrier et la fenetre ovale. *Acta Otolaryngol.*, 16:15, 1931.

New acoustic test, aiming especially at the discovery of an ankylosis between the stapes and the oval window: The new test is of value on account of its constant behavior in otosclerosis, an affection considered by most otologists as the typical form of ankylosis between the oval window and the stapes. (13)

FELDSTEIN, E.: Le traitement chirurgical de la surdité par otospongiose. *Revue gen. de clin. et de therap.*, 46:377, 1932.

The surgical treatment of deafness due to otosclerosis. French text. (14)

FERRERI, G.: La sordità da allatamento. *Acta Soc. Otol. lat. Conv.*, 2:137, 1933.

Deafness due to lactation: Explained as a trophoneurosis. Ear noises and hardness of hearing between the fourth and sixth month of suckling are referred to a trophoneurosis in the domain of the middle and internal ear.

FIALOVSZKY, B.: Otosklerose und Schwangerschaft. *Zentralbl. f. Hals- Nasen- u. Ohrenhlk.*, 22:636.

Hungarian original, German abstract. Circular inquiry in Hungary. Damage sustained in the first pregnancy recedes again in most cases, while that through later pregnancies usually persists. Otosclerosis can never be an absolute indication for the interruption of pregnancy.

FISHBERG, E.: Recent advances in application of biochemistry to otology. *Laryngoscope*, 42:766, 1932.

Includes findings in otosclerosis. "Definite facts for the use of the practising otologist except in the chemistry of the spinal fluid, have been few, but further work along the lines of general calcium and lipoid metabolism are the most promising field for future work."

FLURIN, H., and VIALLE, J.: Indications creno- et climatotherapiques en otorhinolaryngologie. *Arch. internat. de Laryngol.*, 9:129; 257, 1930.

Creno- and climatotherapeutic indications in otorhinolaryngology. Hydromineral medication (crenotherapy) aims in one case at strengthening the

defence processes of the organism, and in another case, on the contrary, at moderating those processes which might become injurious through excess. Bibliography.

FOWLER, E. P.: Calcium, phosphorus and cholesterol in otosclerosis. *Trans. Amer. Otol. Soc.*, 20:39, 1930.

No essential change of the calcium-phosphorus content or of the cholesterol content could be demonstrated in the course of investigations in manifest otosclerosis. In incipient otosclerosis, however, changes may be encountered and careful examination is indicated in all young patients. (9)

—— Otosclerosis, complicated by other lesions. A study of roentgenograms, audiograms, laboratory and clinical findings. *Ann. of Otol., Rhinol. and Laryngol.*, 42:714, 1933.

A study of Roentgenograms, audiograms, laboratory and clinical findings in 126 cases of otosclerosis; 39 cases were studied by microscopic sections in complete series. The clinical evidence indicates that in these cases of otosclerosis there was definite evidence of the almost universal prevalence of past inflammatory lesions in the head spaces, particularly in the nose and middle ear. Examiners should keep in mind the fact that otosclerosis may and usually does occur coincidentally with other lesions of the ear. Illustrated, 3 Figs. (5)

—— Variations in hearing in otosclerosis. *Proc. Amer. Fed. Org. for Hard of Hearing*, 14:11, 1933.

—— Ear conditions from the preventive viewpoint. *Health Examiner*, 5:7, 1935.

Advice in behalf of preservation of hearing and marriage of otosclerotics. Detection of symptoms. Hearing tests. Diagnosis. Treatment. Complications of middle ear disease. Hearing aids. Lip reading. Psychology. (12)

—— The medical aspects of hearing conservation in the New York Schools. *Laryngoscope*, 45:435, 1935.

FOWLER, E. P., JR.: Important points in four theories of otosclerosis. *Trans. Amer. Otol. Soc.*, 21:38, 1931.

Brühl believes that otosclerosis is caused by the irritation of the several factors working at the "otosclerotic corner." Wittmaack believes it to be caused by venous stasis. Weber finds it similar to osteitis fibrosa and puts it on a local metabolic basis. Mayer believes it to be a response to a profound strain, the nature of which he has not yet determined, but the presence of of which he proves by finding of fractures in the points of predilection for the disease.

—— Otosclerosis in ultraviolet light. *Ann. of Otol., Rhinol. and Laryngol.*, 43: 408, 1934.

In ultraviolet light the fiber structure of otosclerotic foci shows it to be a new bone, often as primitive in type as the bone of the normal enchondral capsule. The first bone formed is similar to that of a healing process in any other bone of the body which has been injured, irritated by infections or resorbed by interference with its blood supply. 7 Figs. (13)

—— Suppuration of the petrous pyramid. *Ann. of Otol., Rhinol. and Laryngol.*, 44:1056, 1935.

Petrositis is defined and described as it occurs in portions of the temporal bone. The petrous pyramid is difficult to study by x-ray because of its depth within the skull and because of variations in its own anatomy. 8 Figs. (4)

FRANCIS, R.: Contributions from Sydney, Australia. *Communication, 2 Congrès International d'Otorhinolaryngol., Madrid,* 1932, p. 172.

Reference is made to two cases operated upon by Sourdille, with good results and improved hearing. "In the second case, the ear not surgically treated shows the usual retrogression of otosclerotic progress."

FRANK, I.: The deafness of Beethoven. *Ann. of Otol., Rhinol. and Laryngol.,* 44: 327, 1935.

FRASER, J. S.: The clinical aspects of otosclerosis. *Trans. Internat. Congress of Otorhinolaryngol.,* 1:59, 1932.

Circular inquiries, addressed to specialists, showed the following results: Heredity was stated in 44.4 per cent. In the female line, the disease was transmitted in 79 per cent; in the male line in only 3 per cent. Osteopsathyrosis and blue sclerotics were noted in 24 per cent. Paracusis in 55 per cent. Thyroid and parathyroid medication helped in some instances. The Zund-Burguet electrophonoid was helpful in 90 per cent. (13)

—— Bilateral otosclerosis: radical mastoid operation on worse hearing ear; bony cap of lateral canal removed and skin graft applied. Hearing result seventeen years later. *Scottish Otol. and Laryngol. Soc., Edinburgh, Nov., 1934. J. Laryngol. and Otol.,* 50:700, 1935.

Young woman complaining of deafness in both ears, left worse, of four years' duration; right ear deaf about one year; some tinnitus. Several deaf members in family. Diagnosis, otosclerosis. Operative procedure as stated in superscription. Half a year later, hearing much improved in left ear; tinnitus lessened. Right ear practically the same as before operation. Fistula symptom present on left side.

FREMEL, F.: Wie erkennt und behandelt man die Otosklerose? *Wiener klin. Wchnschr.,* 43:917, 1930.

How does one recognize and treat otosclerosis? In typical cases and in the presence of all the signs of otosclerosis, the diagnosis is rendered without difficulty. In atypical cases with a late onset, absence of the characteristic anamnesis, etc., the distinction from chronic catarrhal conditions and chronic adhesive processes is difficult. A therapeutic trial must here decide; the case is treated for several weeks like a chronic catarrh, and an objectively demonstrable improvement of hearing will exclude otosclerosis.

FRENZEL-SCHEIFF: Zur U-Strahlenbehandlung der Schwerhörigkeit. *Zeitschr. f. Hals-, Nasen- u. Ohrenhlk.,* 31:390, 1932.

Ultra-rays treatment of hardness of hearing: With respect to the U-radiator, an electric-acoustic sound sender, it must be left an open question if biological effects determining a curative effect of any kind are at all possible. (5)

FRIEDBERG, C. K.: Zur Kenntnis des vererbbaren Syndroms: abnorme Knochenbrüchigkeit, blaue Skleren und Schwerhörigkeit. *Klin. Wchnschr.,* 10:830, 1931. *Zeitschr. f. d. ges. Anat.* (Pt. 2), 15:779, 1931.

Contribution to the knowledge of the inheritable syndrome: abnormal bony fragility, blue sclerotics and hardness of hearing. Four out of five cases showed the complete syndrome, including deafness. (4)

GALE, C. K.; JAFFE, H. L.: Labyrinthine capsule in guinea pigs with hyperparathyroidism. *Arch. Otolaryngol.,* 14:815, 1931.

The labyrinth capsules of young guinea pigs, being regions of inactive bone growth, do not show the effects of hyperparathyroidization. Three Figs. (11)

GILSE, P. H. G.: Knochenabbau und Knochenumbau in Felsenbein. *Zentralbl. f. Hals-, Nasen- u. Ohrenhlk.*, 22:709, 1934.

Bone decomposition and bone transformation in the petrous bone: Demonstration of specimens at fifty-fifth meeting of Dutch otolaryngologists, Amsterdam, May, 1933. Besides other factors, differences in pressure and tension perhaps play a part in this connection. (4)

—— Sur la regeneration osseuse de la capsule labyrinthique et de la bulle osseuse chez le chat. *Acta Otolaryngol.*, 20:206, 1934.

Bony regeneration of the labyrinth capsule and the bulla ossea in cats. French text. (11)

GOERKE, M.: Zur Frage der Schwangerschaftsunterbrechung bei Otosklerose. *Folia Otolaryngol.*, Pt. 1, (Orig.) 23:85, 1932.

Interruption of pregnancy in otosclerosis: Based on a lecture held at the 45th meeting of South-Eastern German Otolaryngologists in 1932. Not unconditionally rejected in all cases. (1)

GOLDINBERG, M.: Ein seltener Befund im Knochen der Innenohrkapsel bei kongenitaler Taubheit. *Monatsschr. f. Ohrenhlk.*, 63:35, 1929.

Unusual findings in the bone of the inner ear capsule in congenital deafness: in a congenital deafmute of 35 years, changes of the bone, of otosclerotic type, were found in the environment of the stapes and in the stapes plate itself. Illustrated. 6 Figs. (4)

GOLDSTEIN, M. A.: Problems of the deaf. *St. Louis Laryngoscope Press.* 1933.

Brief paragraph on otosclerosis, in Chapter 13 on the deafened adult.

—— Discussion on otosclerosis. *Trans. Amer. Otol. Soc.*, 20:92, 1930.

GOTTLIEB, M. J.: Contraction of visual fields in cases of progressive deafness. *Arch. Otolaryngol.*, 8:12, 1928.

Report of cases and a comparison with cases of deafness of other types. It is possible that contraction of visual fields does not occur in all cases of progressive deafness. 10 Figures. (13)

—— Constitutional deafness. *Laryngoscope*, 39:485, 1929.

An analysis of cases. Seven charts. (13)

—— A contribution to the etiology of progressive deafness. *Laryngoscope*, 40:85, 1930.

Preliminary report from the Research Clinic on Deafness, Department of Otolaryngology, Beth Israel Hospital, New York. Study of the pancreatic function in nine cases of otosclerosis. Three charts. (8)

—— Deafness due to pancreatic insufficiency. Symptomatology and treatment. *Trans. Amer. Acad. Ophthalmol.*, (1930) 35:385, 1931.

Pancreatic insufficiency is encountered in a large percentage of cases of non-suppurative deafness. That deafness is definitely due to the pancreatic insufficiency is demonstrated by the fact that under suitable replacement therapy, the hearing improves and when the treatment is discontinued, the hearing deteriorates. No matter how profound the deafness, if it is due to pancreatic insufficiency, it can be improved.

—— Deafness, hepatic dysfunction and pancreatic insufficiency; a clinical entity. *Ann. of Otol., Rhinol. and Laryngol.*, 41:523, 1932.

Description of a condition of "auro-hepatic-pancreatic syndrome." The positive objective findings are lowered basal metabolic rate, increase in the number of small lymphocytes in the blood, increased indican in the urine and a definite abnormality in the duodenal fluid. In addition to this, there is a reduction in the digestive activity of the pancreatic ferments. (15)

—— Constitutional deafness. *Arch. Otolaryngol.*, 16:814, 1932.

In this article free use has been made of material and text from "Otosclerosis," 1928. Discussion of predisposing and exciting causes.

—— Constitutional Deafness. *Ann. of Otol., Rhinol. and Laryngol.*, 43:541, 1934.

Constitutional deafness is bilateral, progressive, may begin at any age, shows an aggravation at puberty and in pregnancy, and is caused by toxic and nutritional disturbances. The majority of the cases show a diminished basal metabolism. (9)

GRABSCHEID, E.: Einseitige (?) Otosklerose. Verschlechterung des Hörvermögens nach der siebenten Schwangerschaft. *Zentralbl. f. Hals- Nasen- u. Ohrenhlk.*, 24:718, 1935.

Unilateral (?) otosclerosis. Aggravation of hearing after the seventh pregnancy. The second and third child show otosclerosis, unilateral in the case of the second, bilateral in the third. (1)

GRADENIGO, G.: Sulla eredita morbosa nell' uomo, in particolare riguardo alla sordita familiare. *Riforma Medica*, 40:769, 1924.

Pathological heredity in man, with special reference to family deafness. Italian text. (1)

GRAHE, K., and GRIEBEL, C.: Die Bedeutung der allgemeinen Stoffwechselstörung bei der Otosklerose. *Folia Otolaryngol.*, 22:162, 1932.

The significance of the general metabolic disturbance in otosclerosis. Findings indicate that there is primarily a change of the blood serum in otosclerosis, that gives rise to an increased calcium break-down of the labyrinth bone. Four tabulations. (9)

—— Weitere Abbaustudien bei Otosklerose. *Folia Otolaryngol.*, Pt. 1. (Orig.) 24:149, 1933.

Further reduction studies on otosclerosis: Effect of the urine of otosclerotic and normal persons on the phosphorus content of temporal bones exposed to it for twenty-four hours. Previous investigations had shown that the blood serum of otosclerotics has the capacity of specifically attacking the normal labyrinth bone, showing that otosclerosis does not primarily represent a labyrinth disturbance but a general disease. (9)

GRAIN, R.: L'ionisation iodée en otologie et en laryngologie. *Revue de med.*, 50:646. 1933.

Iodine ionization in otology and laryngology. Local ionization permits the application of more iodine to the seat of the disease than is possible by way of the mouth. In otosclerosis caution is indicated and the method is contra-indicated in the presence of tinnitus or of vertigo.

GRAY, ALBERT A.: Pathological changes in the auditory nerve in otosclerosis and their significance clinically, especially with regard to paracusis Willisii. *Proc. Roy. Soc. Med.*, 25:1286, 1932. *J. Laryngol. and Otol.*, 47:598, 1932.

In otosclerosis there is a degeneration of the fibres of the cochlear nerve, which appears first in the medullary sheath and neurilemma and later in the axis cylinder. (7)

—— Two cases of acusticus tumor with clinical and pathological reports. *J. Laryngol. and Otol.*, 48:535, 1933.

In all such cases, the spiral ganglion is involved and this change occurs at an early date. It is noteworthy that in spite of advanced degenerative changes in the ganglion, Corti's organ remains relatively intact. 9 Figs. (7)

—— The otosclerosis problem; including two cases pathologically examined. *J. Laryngol. and Otol.*, 49:629, 1934.

The essential causative factor of otosclerosis is a gradually increasing defect in the vasomotor mechanism which governs the nutrition of the structure of the organ of hearing as a whole. Eight Figures. (6)

—— The otosclerosis problem; including two cases pathologically examined. *Proc. Roy. Soc. Med.*, 27:1179, 1934.

Dalby Memorial Lecture: The defect in the vasomotor system of the organ of hearing is innate in the individual and not the result of any unfavorable environment. The essential factor without which the disease cannot come into existence is innate in the individual and is therefore, a heritable quality and liable to be transmitted to the offspring. 8 Figs. (6)

—— The causative factor in otosclerosis. *Brit. Med. J.*, 1:868, 1934.

Society report. On the basis of an experience of thirty years and the assembly of the pathological and clinical facts, a case has been made out for explaining otosclerosis by a failure of the vasomotor reflex function of the organ of hearing. (6)

—— The treatment of otosclerotic and similar types of deafness by the local application of thyroxine. *J. Laryngol. and Otol.*, 50:729, 1935.

A large proportion (about 50 per cent) of cases of otosclerosis and so-called dry middle ear catarrh can be greatly improved in regard to both hearing and tinnitus by the intratympanic injection of thyroxine. In seven of fourteen cases treated in this manner, very considerable improvement was obtained. (15)

—— The treatment of otosclerotic and similar types of deafness by the local application of thyroxine. *Laryngoscope*, 45:741, 1935.

"The rationale of the treatment depends upon the writer's view that otosclerosis is the result of a diminished blood supply to the organ of hearing, consequent upon a gradual failure of the vasomotor responses. The action of thyroxine applied locally is to produce an active congestion without inflammatory reaction and continuing for a long period of time." (15)

GRAY, A. A.: Recent work on otosclerosis. *Practitioner*, 135:609, 1935.

GREIFENSTEIN, A.: Vergleichende Untersuchungen zur Histologie der Otosklerose. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 139:14, 1935.

Extensive and detailed histologic investigation of predominantly comparative pathologico-anatomical character led to the result that the focal disease of the labyrinth capsule does not represent a unique bone change. Essentially identical microscopic findings can be noted in bone regeneration and in structural alteration processes of the most variegated kind, which histologically run their course under the picture of osteodystrophic fibrosis. (4)

GRUENBERG, K.: Zur Pathogenese der Otosklerose. *Zeitschr. f. Laryngol.*, 15:78, 1926-7.

Pathogenesis of otosclerosis. Accidental findings in the petrous bone of a man of 23 years with unilateral chronic middle ear suppuration who died from general tuberculosis. The middle ear showed a small active otosclerotic process in the promontory at the lateral insertion of the round window membrane, and also bone proliferations at the capsule of individual semicircular canal segments, interpreted by G. as a developmental anomaly. Five illustrations. (4)

GUGGENHEIM, L. K.: Histopathology and pathogenesis of chronic deafness. *Trans. Amer. Laryngol. Rhinol. and Otol. Soc.*, 37:205, 1931. (3)

—— Phylogenesis and ontogenesis of footplate of stapes, with theory of otosclerosis. *Laryngoscope*, 41:623, 1931.

Preliminary Report of Study: May we not look upon otosclerosis as a regression rather than as a disease process? Nine figures. (3)

—— The development of the organ of hearing. *Ann. of Otol., Rhinol. and Laryngol.*, 40:812, 1931.

—— The cause of otosclerosis. *Ann. of Otol., Rhinol. and Laryngol.*, 41:903, 1932.

Introduction. First of a series of articles by the author of the regression theory. (3)

—— The cause of otosclerosis: Ontogenesis of aural capsule. *Ann. of Otol., Rhinol. and Laryngol.*, 42:251, 596, 1933.

Summary of the development of the mammalian ear (in the human embryo), relating it to the phylogenesis of the ear, and to otosclerosis as a tendency to regression to lower vertebrate types. (3)

—— Otosclerosis: A Regression? *Trans. Amer. Otol. Soc.*, 23:75, 1933.

Discussion: E. Amberg; J. Gordon Wilson. (3)

—— The cause of otosclerosis; the phylogenesis of the ear. *Ann. of Otol., Rhinol. and Laryngol.*, 42:135, 1933.

A discussion of the phylogenesis of the ear from the lowest vertebrates through the birds, with special emphasis on the development of the stapes, oval and round windows, and the relation of this development to otosclerosis in man, as a "regressive dystrophy." (3)

—— Otosclerosis in a seven months' fetus. *Ann. of Otol., Rhinol. and Laryngol.*, 42:690, 1933.

Brief preliminary report: Otosclerotic focus in a seven months' fetus; the earliest reported case. (3)

—— The regression theory of otosclerosis. *Laryngoscope*, 44:499, 1934.

Assumption of otosclerosis as due to the presence in the human ear of fish genes which produce web bone in an attempt to form a capsule of their own period. (3)

—— Otosclerosis. (book), illustrated, 212 pp., St. Louis, 1935.

Introduction covering general consideration of the problem; the various theories of otosclerosis, with critique. The author's theory of regression. The evolution of the ear from the lowest vertebrate to man. The persisting mesenchymal cells retain a potentiality for differentiation and in otosclerosis are activated by the regressive trait of forming a capsule as it is found in lower vertebrates with no oval or round window and no cochlea. The development of the otic capsule is traced from the condensation of mesenchyme about the auditory vesicle at four weeks to its completion.

GUILD, S. R.: Early stages of otosclerosis. *Arch. Otolaryngol.*, 12:457, 1930.

Report of eight cases. Otosclerotic changes may begin as early as the end of the first year of post-natal life. The exact location of the first appearance of the area near the anterior margin of the oval window is not the same in all cases. 27 Figures. (2; 4)

—— The interpretation of audiograms. *Trans. Amer. Otol. Soc.*, 23:91, 1933.

Diagnosis cannot be rendered on the exclusive basis of audiograms. Difficulties arise in differentiating "by means of the functional data" between otosclerosis and certain other conduction lesions. (13)

GUNDRUM, L. K.: Deafness of the child. Report of cases. *Calif. and West. Med.*, 31:134, 1930.

Precise measurements have shown that the removal of tonsils and adenoids was beneficial to hearing, the improvement being more marked than suspected. The best way to "cure deafness" is to prevent it. (12)

—— Influence of treatment on deafness in children. *Laryngoscope*, 43:565, 1933.

"From this study it would seem that the average prognosis for all types is about 75 per cent favorable. The measurement of the hearing of all school children is a goal which we should strive to attain. In this manner many cases are recognized early and if treatment is instituted promptly, much of the deafness in adults can be prevented." (12)

GUTTMANN, M. R., and KULVIN, M.: Value of medical diathermy in certain types of deafness and tinnitus. *Illinois Med. J.*, 60:405, 1931.

Forty-two cases of hardness of hearing were subjected to diathermy treatment, with negative results. The value of diathermy in the treatment of deafness and tinnitus must as yet be proven. (15)

GYLLENSVARD, G.: Osteogenesis imperfecta und blaue Sklera. *Acta Pediatrica*, 13:111, 1932.

Osteogenesis imperfecta and blue sclerotics: Report of a case in a girl of ten years, with fractures demonstrable at birth and occurring later on even during sleep. Brittle bones and blue sclerotics are not unconditionally associated. Early deafness has been observed in families with simultaneous bony fragility. 1 Fig.

HAAG, S.: Ueber den Bau der Labyrinthkapsel einiger Affenarten. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 136:14, 1933.

The normal histological structure of the labyrinth capsule in some simian species; very similar to the human. No direct mention of otosclerosis.

HAFNER, K.: Die knöcherne Labyrinthkapsel der Nagetiere. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 137:282, 1933.

The bony labyrinth capsule of rodents (rabbits, guinea pigs, mice and rats): The osseous capsule of the rabbit and guinea pig has many points in common with the human labyrinth capsule, with regard to its development as well as its mature fibrillar structure. (11)

HAGENS, E. W.: Otosclerosis. *Arch. Otolaryngol.*, 12:273, 1930.

The material for this report consists of four temporal bones from three cases of otosclerosis. Continued study of otosclerosis should be carried out from all available sources. With a good increase in number of temporal bones sectioned, the missing phases of the condition may be found. 8 Figs. (4)

—— Otosclerosis: Staining and chemical reactions. *Arch. Otolaryngol.*, 13:824, 1931.

Material of four temporal bones showing otosclerosis. It could not be proved whether calcium as such was present or absent in the foci. Apparently there is no chemical test by which calcium can be accurately demonstrated. The blue reaction in the foci stained with hematoxylin-eosin may be due to iron. (4)

——— Anatomy and pathology of the petrous bone. *Arch. Otolaryngol.*, 19:556, 1934.

Based on a study of fifty temporal bones. The anatomy of the temporal bone may vary considerably as regards pneumatization. Approximately 34

per cent of the bones studied showed pneumatic spaces in the petrous tip. In acute suppurative otitis media and simple chronic otitis media, there may be an associated infection of the petrous bone, depending on the degree and extent of pneumatization. 14 Figs.

—— Pathology of otosclerosis, with report of cases. *Arch. Otolaryngol.*, 21:297, 1935.

A study of the five examined temporal bones did not reveal any of the pathologic changes in the eighth nerve as described by Gray. The neurilemma was as definite in the cochlear division as in the vestibular. (4)

HALASZ, G.: Otosclerosis and thymus. *Gyogyaszat*, 68:668, 1929.

Hungarian periodical and text. The otosclerosis problem is here discussed from the viewpoint of the internal secretions. Based upon theoretical considerations. H. concludes that a thymus preparation should be employed intravenously in otosclerotic patients. His results were so encouraging that the employment of thymus medication is recommended on a larger clinical material.

HALLPIKE, B. S.: Some observations on bone conduction. *J. Laryngol. and Otol.*, 45:1, 1930.

The importance of the absolute bone conduction test in revealing the loss of cochlear sensibility is stressed. It is suggested that latent cochlear loss in the early stages of chronic middle ear deafness is the most important factor in guiding prognosis and treatment. (13)

HAMM: Eine neue Behandlungsweise chronischer Schwerhörigkeit mit hochfrequenten Schallstrahlen. *Klin. Wchnschr.*, 9:1723, 1930.

A new method of treatment for chronic hardness of hearing with high frequency sound rays. The Mulwert sound radiator represents a great progress in the treatment of chronic hardness of hearing; but in otosclerosis the results are still unsatisfactory. (15)

—— Zur Behandlung der Otosklerose. *Monatsschr. f. Ohrenhlk.*, 66:1313, 1932.

Treatment of otosclerosis. Recommendation of a radiator of Mulwert construction, with high frequency sound waves. This treatment is rather protracted, requiring at least four weeks, but two or three times weekly is sufficient. The prognosis is best in early cases. Report of four benefited cases.

HAMMERSCHLAG, V.: Die Heredopathia acustica und ihr Erbgang. *Wien. med. Wchnschr.*, 82:1019; 1371, 1932.

Faulty acoustic heredity and its transmission: Two lectures on heredity, on the basis of practical illustrative examples. Explanation of simple Mendelian processes. (1)

HARRIS, TH. J.: Contribution to the study of chronic progressive deafness. *Ann. of Otol., Rhinol. and Laryngol.*, 38:661, 1929.

A plea for nation-wide investigation: "Progressive deafness does not have in it the appeal to the popular imagination which tuberculosis has, and yet, with three million school children affected to a greater or less degree, as well as millions more of the adult population with all that it implies of handicap to the wage earner, no greater boon can be brought to mankind than to secure the knowledge by which deafness can be not cured but prevented."

—— The need of a revised nomenclature of chronic progressive deafness. *Trans. Amer. Otol. Soc.*, 23:117, 1933.

The use of the term "Otosclerosis" has only served to increase the difficulty of effecting a satisfactory revision of the nomenclature. The relation of otosclerosis to middle ear disease is a matter of controversy.

HART, V. K.: Bilateral perception deafness of less common etiology. *Laryngoscope*, 41:557, 1931.

Case 3. Atypical otosclerosis.

HELLIN, D.: Treatment of otosclerosis. *Zentralbl. f. Hals- Nasen- u. Ohrenhlk.*, 15:672, 1930.

German abstract of Polish original.

HEYNINX: Y-aurait-il un avantage à tenter la deshypervascularization chirurgicale de la caisse au premier stade congestif de l'otospongiose? *Ann. des mal. de l'oreille*, 43:648, 1924.

Question of eventual advantage of an attempt at surgical relief of the hypervascularization of the antrum in the first congestive stage of otosclerosis.

HINSTORFF, D.: Grundumsatzuntersuchungen bei Otosklerose. University Dissertation, Francfort, 1929. *Zentralbl. f. Hals-, Nasen- u. Ohrenhlk.*, 15:671, 1930.

Investigations of the basal metabolism in otosclerosis. (9)

HIRSCH, G.: Osteodystrophia des Oberkiefers und Otosklerose. *Folia Otolaryngol.*, Pt. 1. (Orig.) 22:438, 1933.

Osteodystrophy of the upper jaw and otosclerosis.

HIRSCHMANN, J.: Untersuchungen ueber das Krankheitsbild der blauen Skleren. *Zeitschr. f. klin. Med.*, 126:718, 1934.

Investigations on the clinical picture of blue sclerotics. Report of six cases in four families. A genealogy of five generations showed a dominant heredity only in the males of the affected families. (4)

HODGSON, H. K. G.: Diagnostic value of x-ray examination of the temporal bone. *Proc. Roy. Soc. Med. (Otol. Sect.)*, 23:717, 1930.

There are cases in which the clinical signs and symptoms are very distinct, and yet the bone changes are very slight. In another case with slight symptoms, the bone changes are very noticeable. In one third of the cases in which there is clinical doubt, H. finds changes which he had only seen associated with otosclerosis. (13)

HOLMGREN, G.: Zur Chirurgie der Otosklerose. *Acta Otolaryngol.*, 15:17, 1931.

Contribution to the surgery of otosclerosis: Permanent results of various personal techniques, all under local anesthesia, were very moderate. Although the tinnitus ceased and the hearing improved immediately after the intervention, the benefit was of brief duration in both. (14)

HOLLENDER, A. R.: Rational uses of physical energies in otorhinolaryngology. *Amer. J. Surg.*, 8:315, 1930.

Postoperative physical aids in otolaryngology: Diathermy with suitable electrodes, zinc ionization, electro-coagulation, ultra-red and ultra-violet irradiation. (15)

HORNICEK, V.: Störungen des humoralen Gleichgewichtes und des vegetativen Nervensystems bei Otosklerose. *Zentralbl. f. Neurologie*, 57:320, 1930-1.

Changes in humoral equilibrium and disorders of the parasympathetic system in otosclerosis. The Hungarian author, in conformity with German and French writers, finds in otosclerosis a diminution of the calcium content in the blood and a lowering of the alkaline reserve. Disturbances in

the vegetative nervous system, seen in some of his cases, are referred to this assumed disturbance in the humoral equilibrium. (9)

HUBBARD, T.: Remarks on the Psychology of deafness. *Laryngoscope*, 32:473, 1922.

"Deprivation of a special sense is a severe test of adaptability. The philosopher meets it by taking stock, as it were, of what is left to him to aid in successfully striving toward the goal, contented well-being. Strength of character is tested, and the real compensation is that the same is fortified by the trials which this handicap entails." (12)

HUGUES, AND OTHERS: Un cas de syndrome de van der Hoeve. *Revue d'otoneuro-ophthal.*, 10:693, 1932.

A case of Van der Hoeve's syndrome: Woman of 38 years, with the full syndrome of blue sclerotics, bony fragility, and deafness, first noted in childhood. Father showed blue sclerotics and deafness; family otherwise negative.

HUNERMANN, T.: Ueber die Anwendung der medizinischen und der chirurgischen Diathermie in der Hals- Nasen- und Ohrenheilkunde. *Zentralbl. f. Hals- Nasen- u. Ohrenhlk.*, 20:417, 1933.

Employment of medical and surgical diathermy in otorhinolaryngology: In otosclerosis in particular, improvement in hearing is reported in a few cases; relief of tinnitus, often temporary, follows in a larger number of cases. (15)

HURTER, H.: Zur Klinik und Symptomatologie der Ostitis deformans Paget. *Arch. f. Ohren-, Nasen- u. Kehlkopfhlk.*, 137:14, 1933.

Incidence and symptomatology of ostitis deformans Paget. Report of six cases, three with advanced and one with incipient deafness of inner ear type. (4)

HUTCHINSON, C. A.: Periotic deafness. *J. R. Army Medical Corps*, 63:296, 1934.

Bony type of otosclerosis: "Proven facts regarding it are: a) that it has a definite racial predilection for Jews; b) that it is especially prevalent in females; c) that in certain cases it certainly is congenital and familial; d) that fragilitas ossium is prevalent in families showing the familial tendency to otosclerosis; e) that a high proportion of cases are congenital in nature, but delayed in onset."

JANUZZI, S. S.: Gruppi sanguigni ed otosclerosi. *Arch. ital. d. otol.*, 40:499, 1929.

Blood groups and otosclerosis. Italian text. The genealogical study of the families of five otosclerotics showed rather an impressive correlation between hereditary transmission of the blood group and hereditary transmission of otosclerosis. Presumably, the blood group is neither cause nor effect, but merely the indicator of a certain general humoral state which to a certain degree is the condition for the development of otosclerosis. 5 Figs.

JELLINEK, A.: Ein Fall von Diplakusia und Beeinflussung des Gehörs durch Lageveränderung. *Monatsschr f. Ohrenhlk.*, 65:985, 1931.

A case of diplacusis and modifications of hearing through a change in position: The patient suffered from otosclerosis with diplacusis and advanced hardness of hearing. (13)

JENKINS, G. J.: Paracusis Willisii and some clinical features of the otosclerosis syndrome. *J. Laryngol. and Otol.*, 47:517, 1932.

Discussion of paracusis as part of the symptom complex of otosclerosis. Assumption of a change in the labyrinth which impairs the auditory acuity in general, more particularly at the lower tone limit. (13)

—— Otosclerosis. *Trans. Internat. Congress Medicine.* Sect. 16 (Otology). Pt. 2, 609.

JOHOW, H.: Ueber die Verwendung des glukonsauren Kalziums in der Hals- Nasen- Ohrenheilkunde. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 126:94, 1930.

Employment of calcium gluconate in otorhinolaryngology: The essential advantage of calcium Sandroz is that in spite of the high calcium contents, it can be injected not only intravenously but also intramuscularly. Calcium therapy is indispensable in otolaryngology; in pathological conditions of allergic character. (15)

JOSEPHSON, E. M.: Vascular changes in chronic progressive deafness. *Laryngoscope*, 39:40, 1929.

In a large series of cases of progressive deafness other than conduction deafness, he observed a characteristic and constant injection of the vessels of the drum and of the inner end of the external auditory canal. (5)

KAHAN, D. S.: L'importance des vitamines dans la clinique de l'otosclerose. *Revue de laryngol.*, 55:1171, 1934.

Importance of vitamins in otosclerosis. Vitamin treatment in the form of ultra-violet irradiation, dry yeast. cod liver oil, etc., led to some improvement in selected cases. (15)

KAVALERCIK, L.: Ein Therapievresuch bei Otosklerose mit einer biologischen Methode. *Zentralbl. f. Hals-, Nasen- u. Ohrenhlk.*, 18-721, 1933.

Tentative therapy of otosclerosis with a biological method: Autohemotherapy in six cases; complete disapparance of the tinnitus in one case; diminution in two cases. (15)

KEELER, J. C.: Modern Otology. Part 10. Otosclerosis and chronic progressive deafness. (book) *F. A. Davis Co.*, Philadelphia, 1930. Chapter 37, p. 689.

Some factors in otosclerosis. The hereditary factor. Endocrine disturbances as a causative factor. Diagnosis. "We do not know whether it is a disease entity, or merely a special symptom of some supposedly correlated condition."

KEEN, I. A.: Clinical observations on chronic deafness in children. *J. Laryngol. and Otol.*, 49:782, 1934.

Clinical histories of thirty-two cases, including a group of otosclerosis. Records of the hearing capacity in the form of graphs. The growing tendency of the diagnosis of otosclerosis is discussed. 32 Figures. (13)

KELEMEN, G.: Ueber die Fissuren im knöchernen Innenohr. *Arch. f. Ohren,- Nasen- u. Kehlkopfhlk.*, 137:36, 1933.

Fissures in the bony internal ear. The microscopically demonstrable fissures in the bone of the inner ear capsule, shown in the petrous bone of adults, have an extensive regularity in their formation. The genesis of the gap is probably due to excessive mechanical taxation, of definite recurrent type. (4, 10)

KERSLEY, G. D.: Brittle bones, blue sclerotics and deafness. *St. Barth. Hosp. Journ.*, 38:236, 1931.

KINDLER, W.: Klinischer Beitrag über das Syndrom Schwerhörigkeit, blaue Skleren, Knochenbrüchigkeit. *Passow-Schaefer's Beitr. z. Anat. Phys. Path. u. Therap. des Ohres*, 30:73, 1932.

Clinical contribution to the syndrome of hardness of hearing, blue sclerotics, bony fragility. Report of illustrative case in a girl of nineteen years. No faulty heredity could be demonstrated. Organic inferiority due

to constitutional factors is assumed as responsible for the origin of the disease. (4)

KOBRAK, H.: Ueber die Beurteilung subjektiver Vestibularissymptome. *Zeitschr. f. Hals- Nasen- u. Ohrenblk.*, 32:386, 1932.

Evaluation of subjective vestibular symptoms. Emphasizes diagnostic difficulties on the basis of an illustrative clinical case. (6, 13)

KOBRAK, H., LINDSAY, J. R., and PERLMAN, H. B.: Investigations on bone conduction in the animal and in the human. *Laryngoscope*, 45:657, 1935.

In the rabbit, there exists an acoustic tensor- and stapedius reflex, which is referable to bone conduction. Patients were observed who showed a stapedius reflex due to bone conduction. Lesions of the sound conduction apparatus of the laboratory animal (obstruction of the external ear, destruction of the chain of auditory ossicles) induced a distinct diminution or disappearance of the sound reflex through air conduction.

KOHN: Osteopsathyrose. *Zentralbl. f. Hals-, Nasen u. Ohrenblk.*, 23:524, 1934-35. (4)

KOHNO, TOSHIJUKI: Experimentelle Forschungen ueber die Zirkulationsstörungen des inneren Ohres. *Mitt. Med. Akad. Kyoto*, 3:37, Abt. B., 1929.

Experimental investigations on circulatory disturbances of the internal ear: On the basis of his findings in rabbits in which embolisms were produced in the labyrinth through injection of foreign substances into the vertebral artery, K. concludes that the resulting pathological changes agree in many points with the anatomical substrata of senile hardness of hearing.

KNUDSEN, V. O., and JONES, P. H.: Bone conduction. *Trans. Amer. Otol. Soc.*, 20:204, 1930. *Arch. Otolaryngol.*, 13:489, 1931.

Study of bone conduction in ordinary sound-proof rooms, including findings in four cases of otosclerosis. It is advantageous to test hearing in a sound-proof room. Charting the percentage of hearing by bone conduction on the same chart with the air conduction, as suggested by Fowler, is of advantage in rendering the diagnosis. (13)

KOMPANEJETZ, S.: Ueber progressive labyrinthäre Schwerhörigkeit der Jugendlichen. *Folia Otolaryngol. Orient.*, 1:237, 1933.

Juvenile progressive labyrinthine hardness of hearing. Boy of eleven years; recessive heredity. Familial occurrence of hardness of hearing; consanguineous marriage of cousins, both from a family with faulty heredity.

KONIGSFEST, R.: Das Krankheitsbild der Osteopsathyrose, kombiniert mit blauen Skleren und Schwerhörigkeit. *Beitr. z. klin. Chir.*, 150:135, 1930.

The clinical picture of osteopsathyrosis, combined with blue sclerotics and hardness of hearing.

KOPILOWITSCH, E. A., and ZUCKERMANN, M. A.: Erfahrungen mit der Behandlung verschiedener Schwerhörigkeitsformen mittels Hochfrequenzschall. *Arch. f. Ohren-, Nasen- u. Kehlkopfhlk.*, 131:208, 1932.

Experiences with the treatment of various forms of deafness by means of high frequency sound waves (ultra-sound): represents a new curative factor in the treatment of certain forms of bad hearing. Minimal results in patients with otosclerosis. (15)

KRAINZ, W.: Veränderungen der Labyrinthkapsel des Kaninchens bei Acidose. *Zeitschr. f. Hals- Nasen- u. Ohrenblk.*, 34:170, 1933.

Changes of the labyrinth capsule in rabbits with acidosis: The bones of young rabbits with artificially induced acidosis showed changes similar to

those of human rickets. These changes were found in the labyrinth capsule as well as other bones, especially in the bony walls of the semicircular canals: osteoporosis and formation of uncalcified bone. (11)

KULEMEYER, W.: Berühmte Schwerhörige und Taube im Lichte erbbiologischer Betrachung. *Deutsche Sonderschule,* 1:692, 1934.

Famous persons hard of hearing or deaf, considered from the viewpoint of heredity and biology. (12)

LAEDERICH, L., MAMON, H., and BEAUCHESNE, H.: Syndrome de van der Hoeve (fragilité osseuse, sclerotiques bleues, surdité). *Bull. et mém. Soc. med. d. Hop. de Par.,* 48:1086, 1932.

The van der Hoeve syndrome: bony fragility, blue sclerotics, deafness.

LAIGNEL-LAVASTINE: Syndromes sympathiques vasomoteurs cerebraux auriculaires et visuels. *Ann. med. psychol.,* 21:207, 1923.

Auricular and visual cerebral vasomotor sympathetic syndromes. (6)

LANGE, W.: Ueber die Anfänge der otosklerotischen Herderkrankung. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.,* 25:1, 1929.

Beginnings of the otosclerotic focal disease: Material of 45 histologically examined cases of otosclerosis. Illustrated: 14 Figures. (4)

—— Fragestellungen zur Histogenese der Otosklerose. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.,* 34:149, 1933.

Queries about the histogenesis of otosclerosis. The blue mantles are regarded as the preliminary stage of otosclerosis. The fissures described by Mayer are explained as artifacts. (4)

LANNOIS, M., and GAILLARD, H.: Yeux ardoisés et surdité totale. *Ann. d'Otolaryngol.,* 2:141, 1933.

Blue sclerotics and total deafness of otosclerotic type without bony fragility, in a patient whose sister had a similar type of deafness, without blue sclerotics or bony fragility. Negative family history. (4)

LAPSLEY, R. M.: Progressive deafness. *J. Iowa Med. Soc.,* 20:160, 1930.

General outline of the subject. Progressive deafness varies in pathology. Otosclerosis has the larger part of its pathology in the changes of the inner ear and the bony change that fixes the stapes in a more or less immobile condition. The bony changes in the fixation of the stapes and the labyrinth resemble the ones in the joints of the fingers in arthritis deformans.

LASAGNA, F.: Indagini radiografiche della otosclerosi. *Rivista oto-neuro-oftal.,* 8:191, 1931.

Radiographic studies in otosclerosis. Italian text.

—— La terapia fisica nel trattamento delle malattie del naso-golo-orecchio. *Boll. mal. orecchio-golo-naso.,* 50:176, 1932.

Physical therapy in the treatment of diseases of the ear, nose and throat. (15)

—— Alcune considerazioni sulla otosclerosi. *Arch. ital. otol.,* 46:133, 1934.

Some comments on otosclerosis: Mechanical factors undoubtedly possess a very great significance in the origin of otosclerosis. The cartoid pulse is assumed as the cause of otosclerosis as well as of fractures in the vicinity of the apical portion of the pyramid, at the labyrinth windows and in the semicircular canals portion of the labyrinth. A cause of electro-chemical nature is also surmised.

LAST, E.: Die Diathermiebehandlung der Otosklerose. *Med. Klin.,* 24:1623, 1929.

Diathermy treatment of otosclerosis and of chronic affections of the internal ear due to circulatory disturbances. Otodiathermy aims in the

first place at the removal of subjective disturbances and in many cases meets the purpose, when properly managed. (15)

LEALE, G.: Contenuto minerale nel sangue degli otosclerotici. *Valsalva*, 7:77, 1931.

Mineral content of the blood of otosclerotics.

LEICHER, H.: Otorhinolaryngologie und endokrines Drüsensystem. *Klin. Wchnschr.*, 7:2229, 1928.

Otorhinolaryngology and endocrine glandular system. (8; 9)

—— Otosclerosis y secrecion interna. *Rev. med. German-Iber-Amer.*, 2:109, 1929.

Otosclerosis and internal secretion. The relations between otosclerosis and internal secretion are shown in the first place by the frequent manifestation or aggravation of the otic process at the time of puberty, pregnancy, menstruation, menopause. The influence on the production of otosclerosis is referable to certain endocrinic modifications of the blood during the sexual phases. (8)

—— Erbkrankheiten des Hörorgans. *Med. Klin.*, 2:1096, 1935.

Hereditary diseases of the auditory organ. Brief review of modern knowledge on the subject.

LEIDLER, R.: Vestibulärapparat und vegetatives System: Einfluss dieses Systems auf den peripheren Vestibulärapparat. *Monatsschr. f. Ohrenhlk.*, 68:455, 1934.

Vestibular apparatus and vegetative system: Influence of this system upon the peripheral vestibular apparatus. (6)

—— Vestibulärapparat und vegetatives System: Der Einfluss des Vestibulärapparates auf das vegetative System. *Monatsschr. f. Ohrenhlk.*, 69:1, 1935.

Vestibular apparatus and vegetative system: The influence of the vestibular apparatus upon the vegetative system. (6)

LEIRI, F.: Ueber die Bedeutung der mechanischen Reizung als aetiologisches Moment in der Pathogenese der Otosklerose. *Acta Otolaryngol.*, 13:312, 1929.

Significance of mechanical irritation as an etiological factor in the pathogenesis of otosclerosis. (4, 10)

—— Ueber Aehnlichkeiten in den Krankheitsbildern der Otosklerose und der juvenilen Epiphysenaffektionen. *Acta Otolaryngol.*, 16:57, 1931.

Similarities in the pathological pictures of otosclerosis and juvenile epiphyseal diseases. Bibliography. 3 Figures. (2: 4)

—— Ueber die Knorpelfuge vor dem ovalen Fenster. *Acta Otolaryngol.*, 15:74, 1931.

The cartilage zone in front of the oval window in different stages of development. 5 Figures. This zone is not of vascular origin and vessels are not a condition for its presence. The connective tissue strand in the cartilage zone is presumably a tissue of very different character than the perivascular tissue around the vessels in the semicircular canals portion of the labyrinth. (2)

—— Giebt es einen elektro-chemischen Faktor in der Pathogenese der Otosklerose? *Communication, 2 Congrès International d'Otorhinolaryngologie, Madrid*, 1932. 136.

Is there an electro-chemical factor in the pathogenesis of otosclerosis? This question is answered in the affirmative, with enumeration of data inducing a stronger electrochemical factor in otosclerotic than in normal cases. (13)

—— Pathogenese der Otosklerose. *Zentralbl. f. Hals-, Nasen- u. Ohrenhlk.*, 20:191, 1933.

Pathogenesis of otosclerosis. Certain mechanical irritants and electro-

chemical factors are held responsible for the production of the otosclerotic changes in the labyrinth capsule. (4)

—— Ueber die Pathogenese der Otosklerose. *Acta Otolaryngol.*, 19:427, 1934.

Pathogenesis of otosclerosis. Mechanical factors, electro-chemical factors. The localization of the otosclerotic foci in the vicinity of the mechanically most strongly affected portions of the labyrinth capsule indicates that the mechanical stimulus is responsible. (10)

LENART, G.: Die Nebenschilddrüsenfunktion. *Ergebnisse der Inneren Medizin*, 46: 446, 1934.

Extensive discussion of the parathyroid function. In otosclerosis, internal secretory relations are probable and an eventual hypoparathyroid genesis is suggested by the diminution of the blood calcium in the majority of the cases. (8)

LERMOYEZ, M.: Le vertige qui fait entendre. *Presse méd.*, 27:1, 1919.

Vertigo that restores hearing. (6)

LEWIS, E. R.: Otosclerosis as an anachronistic phenomenon. *Trans. Amer. Otol. Soc.*, 22:318, 1932.

Otosclerotic bone changes show reversions to immature and embryonal characteristics, among which is the devotion of cell activities almost exclusively to growth; in this respect otosclerosis might be described as anachronistic rather than pathologic. (4)

—— Improved hearing in fixation deafness. *Ann. of Otol., Rhinol. and Laryngol.*, 42:135, 1933.

Discussion of otosclerosis as a change in the structure of the bone cells, resulting from biological and biochemical factors. Report of two cases of progressive deafness improved by systemic treatment designed to alter "the constitutional body tissues." (4)

LEWY, A.: Influence of fluorine on the bony labyrinth of the white mouse. *Arch. Otolaryngol.*, 8:315, 1928.

Preliminary report. Fluorine is a factor in bone metabolism to be reckoned with, and in the white mouse its influence appears to extend to the bony labyrinth of the ear. To the otologist it suggests the possibility of its application in otosclerosis. (11)

—— Influence of fluorine on the bony labyrinth of the white mouse. Further observations. *Arch. Otolaryngol.*, 20:693, 1934.

Changes of the labyrinth bone, consisting of a persisting immaturity, could be shown in some of the laboratory animals. (11)

LICHATSCHEFF, A.: Treatment of some diseases of the ear, nose and throat with mineral mud packs. *Revue de laryngol., otol., et rhinol.*, 55:263, 1934.

French abstract of Russian original. Ear diseases are treated by the application of warm mineral mud packs, one placed over the ear after closing the external meatus by a tampon, and one over the mastoid process. Excellent results are claimed in chronic catarrhal deafness. Only slight if any benefit in otosclerosis.

LLEUWEN, H. C. VAN: Ueber familiäres Vorkommen von Lipodystrophia progressiva zusammen mit Otosklerose, Knochencysten und geistiger Debilität. *Zeitschr. f. klin. Med.*, 123:534, 1933.

Familial occurrence of progressive lipodystrophy together with otosclerosis, bone cysts and mental debility: Three cases in sisters. The parents and other children were normal. Possibility of recessive hereditary factors. (4)

LLOPIS-LLORET, B.: Sobre las reaciones paranoides de los sordos. *Archives de Neurobiol.*, 13:1117, 1933.

Paranoiac reactions of the deaf. Spanish text. (12)

LOEBELL and NAUCK: Die funktionelle Anatomie des menschlichen Felsenbeins. *Zeitschr. f. Hals-, Nasen- u. Ohrenhlk.*, 34:129, 1933.

The functional anatomy of the human petrous bone: Anatomical studies of various portions of the temporal bone in relation to function; twenty-seven specimens, two embryonic, of the labyrinth capsule. Demonstration of Mayer's fissures. (4)

LOEWENSTEIN, W.: Kann durch Knochentransplantation der Blutkalkspiegel des Menschen erhöht werden? *Monatsschr. f. Ohrenhlk.*, 68:1023, 1934.

Contribution to the treatment of otosclerosis by transplantation of bone: Can the blood calcium level in man be raised through bone transplantation? Grafts under the subcutaneous cellular tissue of the anterior thoracic wall, in eleven cases of otosclerosis, induced no increase of the blood calcium and no modification of the otosclerosis was demonstrable. No lasting improvement of hearing. (14, 15)

MAFFEO, L.: Il fattore Mendeliano nella otosclerosi. *Il Morgagni*, 72:1414, 1930.

The Mendelian factor in otosclerosis: Synthetic review. Auricular sclerosis (chronic catarrhal otitis media) and otosclerosis are often confused. More extensive investigations may permit positively to ascertain otosclerosis as a familial disease and as such transmissible in the generations according to the Mendelian type. (1)

MAGNOTTI, T.: L'ovaio in rapporto all'ipofisi ed all'otosclerosi. *Valsalva*, 8:769, 1932.

The ovary in relation to the hypophysis and to otosclerosis: Sclerotic changes were found in the labyrinth capsule in young rabbits after castration and are explained by hyperfunction of the pituitary resulting from lack of ovarian hormone. (8)

MALECKI, J.: Diagnostic value of the hydrogen ions concentration and alkaline reserve in otosclerosis. *Polska Przeglad Oto-Laryngol.*, 9:138, 1935.

In nine of eleven examined cases, the acid-base equilibrium, which is normal in other forms of chronic progressive deafness, was displaced in the acid direction. An alkaline reserve below 50 suggests the diagnosis of otosclerosis.

MALHERBE, A.: Technique de l'electro-ionisation transtympanique dans l'otite osteo-spongieuse. *Bull. med.*, 421193, 1928.

Technic of transtympanic electro-ionization in osteosclerotic otitis. (15)

—— Symptomatologie et diagnostic de l'otite osteo-spongieuse. *Presse med.*, 38: 237, 1930.

Symptomatology and diagnosis of osteosclerotic otitis. (13)

MALIUTIN, E. M., and NALETOVA, M. W.: Die progressierende Schwerhörigkeit und ihre Behandlung. *Soviet Klinik*, 20:975, 1934.

Progressive hardness of hearing and its treatment: The treatment of progressive deafness of otosclerotic as well as other origin should consider also the psychic condition of the patients. Special care, more particularly at the age of puberty, is needed for the children of otosclerotic parents.

MANASSE, P.: Neue Untersuchungen zur Otosklerosefrage. *Zeitschr. f. Ohrenhlk.*, 82:76, 1922.

New investigations on the otosclerosis problem.

MARINUS, C. J.: Relation of the endocrines to eye, ear, nose and throat. *Trans. Amer. Acad. Ophthal.*, 38:238, 1933.

MARIETOU: Un cas de maladie de van der Hoeve. *Bull. Soc. d'Ophthal. de Paris*, 6:448, 1933.

A case of van der Hoeve's syndrome: Man of 33 years, with a history of several spontaneous fractures since the age of eleven; blue sclerotics and deafness, most marked in the right ear. The deafness was of the mixed type. Mild hypothyroidism. Negative family history. The observer regards the deafness of fragilitas ossium as otosclerosis with the possibility of involvement of the internal ear.

MAYBAUM, J. B., and DEUSS, J. G.: Geniculate ganglion (Hunt's syndrome). Clinical features and histopathology. *Arch. Otolaryngol.*, 19:574, 1934.

The clinical features and histologic changes in a case of Hunt's syndrome (man of 51 years) are presented. Of additional and unusual interest in this case is the histologic evidence of otosclerosis and bilateral labyrinthitis. (7)

MAYER, O.: Zur Pathogenese und Aetiologie der Otosklerose. *Monatsschr. f. Ohrenhlk.*, 45:421, 1911.

The focal disease of the bone of the pyramid is produced through local circulatory disturbances in the domain of the terminal distributions of the nutrient arteries of the bone. Clinical observations prove the occurrence of vasomotor disturbances in otosclerotics. The responsible circulatory disturbances seem to be primarily referable to disturbances in the function of the vasomotors; but organic vascular affections may also play a part in this connection. (5; 6)

—— Untersuchungen über die Otosklerose. *Monograph*, Vienna, 1917.

Investigations on otosclerosis: Interpretation of the focal change as a special form of tumor-like hyperplasia; to be grouped under the heading of "hamartoma." (4)

—— Ueber einige Streitfragen aus der Knochenhistologie der Labyrinthkapsel mit Beziehung auf die Veränderung der Labyrinthkapsel bei Otosklerose. *Zeitschr. f. Hals-, Nasen- u. Ohrenhlk.*, 9:187, 1924.

Some controversial questions from the bone histology of the labyrinth capsule with regard to the change of the labyrinth capsule in otosclerosis. (4)

—— Pathology of Otosclerosis. Proc. Roy. Soc. Med. (Sect. Otol.), 21:77, 1928. *J. Laryngol. and Otol.*, 43:843, 1928.

"The results of all these investigations lead us to the conclusion that areas of otosclerosis are to be regarded as hyperplasias." "Only on the basis of this view are we able to understand the whole clinical and anatomical picture." (4)

—— Ueber die Entstehung der Spontanfrakturen der Labyrinthkapsel und ihre Bedeutung für die Otosklerose. *Zeitschr. f. Hals-, Nasen- u. Ohrenhlk.*, 26: 261, 1930.

The origin of spontaneous fractures of the labyrinth capsule and their significance for otosclerosis: A special vulnerability of the labyrinth capsule, due to its composition of brittle compact bone, is held responsible for the occurrence of these spontaneous fractures. (2; 4; 10)

—— Die Ursache der Knochenneubildung bei der Otosklerose. *Acta Otolaryngol.*, 15:35; 407, 1931.

The cause of bony new formation in otosclerosis: The presence of spon-

taneous fractures in the labyrinth capsule proves the existence of special tensions, to which the capsule often gives way and breaks. The otosclerotic foci supervene at these points where special capsular tensions exist; hence they are most common in the periosteal zone in front of the oval window. 15 Figures. (4)

—— Die Entstehung der Spontanfrakturen der Labyrinthkapsel und ihre Bedeutung für die Otosklerose. *Zeitschr. f. Hals-, Nasen- u. Ohrenhlk.*, 28:228, 1931.

The origin of spontaneous fractures of the labyrinth capsule and their significance for otosclerosis: Reply to Brühl's comments on Mayer's work (1930). Emphasizes the importance of mechanical taxation of the labyrinth capsule as a part of the entire pyramid. The process of otosclerotic bony new formation through substitution of the lamellar bone by young plexus-like bone is a kind of protective callus formation against mechanical strain of the bony labyrinth capsule. 12 Figures. (4; 10)

—— Vortrag über die Otosklerose. *Monatsschr. f. Ohrenhlk.*, 66:1136, 1932.

Lecture on Otosclerosis, held at the Austrian Otological Society, May, 1932. With discussion. The bony new formation in otosclerosis probably originates in consequence of mechanical-functional taxation and serves to protect those areas of the labyrinth capsule which are especially endangered due to traction and tension, by means of resistant bone instead of the mechanically inferior old bone. The process aims at rectification of structural faults and weakness of the labyrinth capsule. (4; 10)

—— Referat über die pathologische Anatomie und Pathogenese der Otosklerose. *Trans. Internat. Otorhinolaryngol. Congress. Section on Otosclerosis*, Madrid, 1932.

Survey of pathological anatomy and pathogenesis of otosclerosis. The most important guide must remain the study of general bone pathology. The otosclerotic process is not unique and restricted to the labyrinth capsule, but has its analogy in other localities of the osseous system. (4)

MAYOUX, R.: Les indications du traitement de la surdité par la haute fréquence. *Lyon Med.*, 149:661, 1932.

Indications for high frequency treatment of deafness: The vasoconstrictor action of tension applications seems to destine them for the treatment of otosclerosis, but never gave very distinct results. The most beneficial action was observed in some cases of aural tinnitus without noteworthy diminution of hearing. (15)

MCKENZIE, D.: Otosclerosis: A clinical analysis. *J. Laryngol. and Otol.*, 47:92, 1932.

Formulation of a number of conclusions and hypotheses, based upon the study of over 600 cases of otosclerosis from his private practice. These cases are divided into primary, secondary and incipient forms. The development of the disease at any age averages three and a half years, although in some especially grave cases a very marked degree of deafness may supervene very rapidly, whereas in others the hardness of hearing progresses very slowly.

—— Graphs illustrating otosclerosis. *Proc. Roy. Soc. Med.*, 25:483, 1932.

MENDOZA, R.: Las otosclerosis (aspecto clinico). Communication, 2 Congres International d'Otorhinolaryngol., Madrid, 1932. 170.

Otosclerosis; clinical aspect. Study of disturbances of accommodation, in the form of anacusis, paracusis and acusis. The latter is seen in advanced otosclerosis.

MENNINGER, K.: The mental effects of deafness. *Proc. of the Fourth Annual Conference of the Amer. Federation of Organizations for the Hard of Hearing (Incorporated)*, Chicago, June, 1923.

Emotional maladjustment manifests itself in two clinical forms, one characterized by depression, the other by suspicion. (12)

MEYER, M.: Ueber den feineren Bau des Knochengewebes in der normalen menschlichen Labyrinthkapsel. *Zeitschr. f. Hals-, Nasen- u. Ohrenhlk.*, 18:297, 1927-8.

The finer structure of the osseous tissue in the normal human labyrinth capsule.

—— Ueber ein besonderes Knochengewebe in der menschlichen Labyrinthkapsel. *Verhandl. d. phys. med. Ges.*, 52:88, 1927; 57:26, 1932.

A special osseous tissue in the human labyrinth capsule.

—— Beiträge zur normalen und pathologischen Anatomie der Knöchernen Labyrinthkapsel. *Beit. z. Anat. Phys. Path. u. Ther. des Ohres*, 28:357; 157:171; 201, 1931.

Contributions to the normal and pathological anatomy of the bony labyrinth capsule. Four articles, in part dealing with the labyrinth capsule in osteogenesis imperfecta congenita and in rickets and osteomalacia.

—— Patogenesia y terapeutica de la otosclerosis. *Rev. Med. Germ. Iber-Amer.*, 4:25, 1931.

Pathogenesis and therapy of otosclerosis: A discussion of the various theories. Therapeutically, only the associated symptomatic ear noises are more or less amenable to treatment with pyramidon and bromides.

—— Die normale Anatomie der Labyrinthkapsel. *Zitschr. f. Hals- Nasen- u. Ohrenhlk.*, 34:1, 1933.

The normal anatomy of the labyrinth capsule: Investigations on humans and laboratory animals. The hen, on account of the special vascular arrangement of its labyrinth capsule, seems to be more suitable than mammals for experimental investigations.

—— Ueber einige für die Pathologie der Erkrankungen des Skeletsystem interessante Beobachtungen an der knöchernen Innenohrkapsel des Menschen. *Virchow's Arch. f. path. Anat.*, 288:1, 1933.

Some observations on the bony internal ear capsule in man, of interest for the pathology of affections of the skeletal system. 14 Figs.

—— Pathogenese und Therapie der Otosklerose. *Tung-Chi med. Wchnschr.*, 10:123, 1935.

Pathogenesis and therapy of otosclerosis: The etiology being still a matter of controversy, there can be no etiological therapy. The foci in his opinion consist entirely of new formed bone substance with a spongy structure, differing from the original compacta.

MIRVISH, L.: Otosclerosis: a metabolic disorder. *J. Laryngol. and Otol.*, 45:449, 1930.

This work represents an intensive study of a few cases of otosclerosis treated with parathormone injections. The hearing was continuously controlled by quantitative hearing tests. In the reported cases, parathormone administration definitely arrested the progress of the deafness and in two cases improved hearing. (8; 15)

MOELLER, E.: The family tree of a patient with Eddowes' syndrome. *Acta med. Scandinav.*, 69:223, 1928.

The hereditary occurrence of blue sclerotics and bony fragility was

first described by Eddowes in 1900. These two abnormalities were later shown to be frequently combined with otosclerosis. The family tree of this patient, a woman of 29 years, gives support to the supposition that blue sclerotics depend on a simple dominant factor. On the other hand, it is not possible to draw any conclusions about the hereditary causes of the occurrence of bony fragility and deafness in the phenotype.

MONDOR, and SICARD, A.: Un cas d'osteopsathyrose. *Presse med.*, 41:1856, 1933.

A case of osteopsathyrosis: Woman of 50 years, with multiple fractures, blue sclerotics and progressive deafness of the right ear. Family history negative, in so far as known to the patient.

MOULONGUET, A.: Une nouvelle technique chirurgicale pour le traitement de l'otosclérose. *Ann. d'Otolaryngol.*, 1:132, 1932.

A new surgical technic for the treatment of otosclerosis: The author proposes the employment of a palpebral cutaneous flap from the skin of the upper eyelid, to close the trephine orifice in Sourdille's operation. (14)

—— Traitement chirurgical des bourdonnements d'oreille. Etude historique et critique. *Act. Soc. Otol. lat. 3 Conv.*, 1:153; 2:129, 1933.

Surgical treatment of aural tinnitus. Historical and critical study.

—— Traitement chirurgical des bourdonnements d'oreille; Etude historique et critique. *Otologie Internat.*, 18:88, 1934.

Surgical treatment of aural tinnitus. Historical and critical study. Distinction between subjective and objective ear noises; subdivision of the former into noises of vascular and muscular genesis. No cure is guaranteed by surgical interventions. (14)

MOULONGUET, A., and DONIOL, T. L.: Traitement de la surdité par la diathermie. *Ann. des mal. de l'oreille*, 49:909, 1930.

Treatment of deafness by diathermy: Results in a series of diathermic treatments, including five otosclerotic women whose hearing was not improved. Although in three cases, some improvement of the vertigo and tinnitus was noted, other patients could not continue the treatment on account of the onset and increase of these disturbances. (15)

—— Apropos d'un nouveau phenomène acoustique et d'une nouvelle méthode d'acoumetrie. *Ann. d'Otolaryngol.*, no. 3:324, 1931.

A new acoustic phenomenon and a new acoumetric method. (13)

MOUNIER-KUHN, P., and CECCALDI, A.: Essais d'electrotherapie en Otologie. *Revue de laryngol.*, 53:759, 1932.

Electro-therapeutic attempts in otology: Diathermy, ionization, ultra violet rays. Nineteen observations, including nine cases of otosclerosis. Diathermy or ultra violet rays gave zero results in otosclerosis. Ear noises were benefited by ionization. Hypacusis is generally not improved, especially in otosclerotics. (15)

MUCK, O.: Beitrag zum Konstitutionsproblem der Otosklerose. *Zeitschr. f. Hals-Nasen- u. Ohrenhlk.*, 30:375, 1932.

Contribution to the constitutional problem of otosclerosis. According to personal investigations since 1925, the autonomous nervous system must have undergone a disturbance in otosclerosis. The vascular stigmata and the white streak were found to be characteristic of otosclerosis, indicating a disturbance of the tonus in the domain of the internal carotid or the auditory artery. (9)

—— Ueber einen vasomotorisch bedingten Nystagmus und seine Unterscheidung von labyrinthären. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 30:668, 1932.

Nystagmus of vasomotor origin and its differentiation from labyrinthine nystagmus. (6)

—— Nystagmus of vasomotor origin and its differentiation from labyrinthine nystagmus. *Arch. Otolaryngol.*, 17:587, 1933.

English abstract of German article in Zeitschr. f. Hals-, Nasen- u. Ohrenhlk. 30:668, 1932. (6)

—— Ueber den Gesichtsausdrück Schwerhöriger. *Münch. Med. Wchnschr.*, 1:145, 1935.

The facial expression of the hard of hearing. Attentive listening on the part of a person hard of hearing causes relaxation of the body muscles including the small muscles around the mouth. The result is the open mouth seen in many persons hard of hearing. (12)

MULWERT, H., and VOSS, O.: Eine neue physikalische Behandlungsmethode chronischer Schwerhörigkeit und deren Ergebnisse. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 119:81, 1928.

A new physical method of treatment for chronic hardness of hearing and its results. Description of an apparatus serving for production of high frequency sound waves, beyond the upper hearing limit. Improvement of hearing, although alternating with aggravations is obtained also in otosclerosis. 3 Figs. and 6 curves. (15)

MYGIND, S. H.: Die nicht suppurativen Schallleitungsleiden. *Acta Otolaryngol.*, 16:333, 1931.

The non-suppurative impairments of sound conduction. The relation of catarrhal otitis to otosclerosis. Middle ear and labyrinth form an inseparable unit and react in the same manner to a great variety of injurious factors. With its twofold acoustic and vestibular function, the ear is an especially delicate indicator of these influences on the state of the vessels, especially the capillaries.

—— Le bourdonnement de l'oreille. *Acta Otolaryngol.*, 15:426, 1931.

Aural tinnitus. This is presumably referable to intracellular edema of Corti's organ, which results in swelling of the capillary and Deiters' cells. In otosclerosis, the tinnitus is referred to stasis in the bone veins.

NAGER, F. R.: Demonstration zur Osteopathologie der Labyrinthkapsel. *Acta Otolaryngol.*, 15:413, 1931.

Demonstration on the osteopathology of the labyrinth capsule: A series of otosclerosis specimens of interest by the combination with other bone diseases. Exclusive involvement of the periosteal labyrinth capsule. (4)

NAGER, F. R., and MEYER, M.: Beiträge zur normalen und pathologischen Histologie der knöchernen Labyrinthkapsel. *Passow-Schaefer's Beitr. z. Anat. Physiol., Pathol. u. Therap. des Ohres*, 28:357; 29:157; 30:23. Illustrated.

Contributions to the normal and pathological histology of the bony labyrinth capsule. Detailed description of the histological findings. (4)

—— Die Erkrankungen des Knochensystems und ihre Erscheinungen an der Innenohrkapsel des Menschen. *Monograph, Berlin, S. Karger*, 1932.

Diseases of the bone system and their manifestations in the human internal ear capsule.

—— Die Stellung der Otosklerose in der heutigen Knochenpathologie. *Zentralbl. f. Hals- Nasen- u. Ohrenhlk.*, 19:74, 1932-33.

Extensive bone studies showed a fundamental histological conformity

between ostitis fibrosa, ostitis deformans, on the one hand, and otosclerosis on the other. This fundamental conformity leads to the grouping of the otosclerotic change of the labyrinth capsule under the heading of osteoses or osteodystrophies, rather than as an isolated phenomenon in bone pathology. (4)

NAGER, F. R.: Die Stellung der Otosklerose in der heutigen Knochenpathologie. *Folia Otolaryngol.*, Pt. 2, (Ref.) 36:73, 1933.

Demonstration, on petrous bone sections, of the extensive histological similarity of ostitis fibrosa Recklinghausen, ostitis deformans Paget and otosclerosis. (4)

—— Die pathologische Anatomie der Labyrinthkapsel und ihre klinische Bedeutung. *Zeitschr. f. Hals-, Nasen- u. Ohrenhlk.*, 34:72, 1933.

The pathological anatomy of the labyrinth capsule and its clinical significance: Summary of the pathologic changes in the labyrinth capsule in various bone diseases. Classification of otosclerosis as a localized fibrous osteodystrophy. (4)

—— Les particularités histologiques et biologiques de la capsule labyrinthique. *Revue de laryngol.*, 56:832, 1935.

The histologic and biological peculiarities of the labyrinth capsule.

NASH, C. S.: A study in otosclerosis. *Ann. of Otol., Rhinol. and Laryngol.*, 39: 769, 1930.

In a series of one thousand cases of deafness, less than six per cent were definitely diagnosed as otosclerosis. A careful histopathologic distinction must be made between a primary otosclerosis and secondary atrophic lesions produced by stapes ankylosis of middle ear origin. (13)

NESFIELD, V. B.: Deafness and its alleviation by operation. *Monograph, London*, 1931. 167 pages.

In otosclerosis the results of operation have not been so good as in cases of deafness caused by middle ear catarrh; except that deafness has been arrested and that there has been decided improvement in hearing so long as the new canal has been kept open enough to allow air to be blown through easily. In no case in his experience has the deafness been made worse. (14)

NEUMANN, H.: Kombinierte Mittelohr-Innenohraffektion; Otosklerose? Lues? *Zentralbl. f. Hals- Nasen- u. Ohrenhlk.*, 20:576, 1933.

Combined middle ear- inner ear affection. Otosclerosis? Lues? Society report. No caloric reaction. Preserved rotatory reaction. Negative Wassermann test. (13)

NEVILLE, W. S. T.: Chronic deafness. *Proc. Roy. Soc. Med.*, 22:1414, 1929.

In a case of otosclerosis in a woman of 39 years, who complained of deafness, tinnitus and giddiness, improvement was obtained by diathermy and Zund-Burguet electrophonoide.—The theory on which the use of diathermy is based is that by producing localized heat in deep tissues, the blood supply is augmented and this leads to the absorption of effusions, softening of exudates and fibrous tissue and relaxation of muscle spasms. (15)

NIKOLAJEW, N. A.: Ueber das Gefässsymptom in der Otologie und seine Bedeutung. *Zentralbl. f. Hals- Nasen- u. Ohrenhlk.*, 24:369, 1935.

The vascular symptom in otology and its significance: Recommendation of a vascular test, as simple and practical for diagnostic purposes. (13; 15)

NOACK, C. L.: Erfahrungen mit Brojosan bei Otosklerose. *Münch. med. Wchnschr.*, 76:580, 1929.

Experience with Brojosan in otosclerosis: Since two years, brojosan tablets (iod. brom.) have been successfully utilized in diseases of the middle and internal ear, and in otosclerosis with its strong subjective aural noises. The treatment must be prolonged (5-6 weeks) and combined with a salt-poor diet. (15)

ÖHNELL, H.: Om scorbutisk Ménière och otoscleros. *Svenska Läkaresällskapets Handlingar*, 57:358, 1931.

Scorbutic Meniére's symptom and otosclerosis: Vitamin C deficiency.

OZAWA, M.: Einfluss des Funktionsausfalls vom Halssympathikus auf das Ohr. *Mitt. Med. Akad. Kioto*, 12:267, 1934.

Experimental studies on the influence of the functional loss of the cervical sympathetic on the ear. Degenerative changes at the nerve end apparatus and the stria vascularis of the labyrinth, in guinea pigs and rabbits. The findings are eventually applicable to man. (11; 6)

PALIARD, P.: De l'importance des troubles endocriniens dans l'otospongiose. *Marseille med.*, 1:440, 1930.

The importance of endocrinic disturbances in otosclerosis: Hyperfunction of the thyroid gland, hypofunction of the ovary, the hypophysis and the suprarenal. Since the recent endocrine discoveries, otosclerosis is no longer to be regarded as an incurable disease. (4; 8)

PARREL, G. de: La surdité familiale et le mariage des sourds. *Presse med.*, 39:67, 1931.

Familial deafness and marriage of deaf persons. Includes a discussion of heredity in otosclerosis.

—— La reéducation chez les otoscléreux. *Communication, 2 Congres International d'otorhinolaryngol.*, Madrid, 1932.

The term "otosclerotic patients" is here applied to cases of bony stapedio-vestibular ankylosis wtih osteosclerosis of the labyrinth capsule. (Psycho-visual re-education, completed by oral acoustic exercises, represents the most efficient means of assistance for otosclerotics. The ear participates in lip-reading, which is not anti-acoustic, but pro-acoustic.) (15)

—— La re-education en los otosclerosos. *Revista Espan. y Amer. de Laryngol. Otol. y Rinol.*, 23:587, 1932.

Re-education of otosclerotics: Instruction in lip-reading is recommended as the best means of re-education in otosclerosis. (15)

—— La question de l'otospongiose. *Clinique*, 27:333, 1933.

The otosclerosis question: A general review of the subject, based largely on the papers presented at the Madrid International Congress.

PENDE, N.: Patologia dell'apparato surrenale e degli organi parasimpatici. *Monograph*, Milan, 1909.

Pathology of the suprarenal apparatus and of the parasympathetic organs. Italian text. Both the Lermoyez and the Ménière syndrome are interpreted as signs of a disturbance in the vegetative nervous system. (6)

PERWITZSCHKY, R.: Zum Problem der Einwirkung ultra-akoustischer Schallwellen auf das schwerhörige Ohr. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 34:237, 1933.

The problem of the action of ultra-acoustic sound waves upon partially deaf ears: Use of ultra high frequency sound waves in the treatment of

various forms of deafness, including forty-six cases of otosclerosis. Definite improvement in one case; doubtful benefit in twenty cases. (15)

—— Ueber den therapeutischen Einfluss kurzer Schallwellen auf das Ohr. *Arch. f. Ohren-, Nasen- u. Kehlkopfhlk.*, 136:124, 1933.

Therapeutic influence of short sound waves upon the ear: Discussion of the theory of the use of ultra high frequency sound waves in the treatment of deafness. Results in deafness of various types. 164 cases, including 21 cases of otosclerosis. (15)

PHILLIPS, WENDELL G.: Conservation of hearing. A health problem in human efficiency. *Volta Review*, 29:639, 1927.

The psychological aspect of acquired deafness is the result of the more or less rapid destruction of the power of free human communication in business, society and even in the street and shop. (12)

PORTA, C. F.: Contributi allo studio della morfologia e della patologia dell'osso temporale. Nota 1: Gli emissari venosi mastoidei. Nota 2: Gli emissari squamosi e petrosquamosi. *L'Otorinolaringol. Italiana*, 5:483; 608, 1935.

Contributions to the study of the morphology and pathology of the temporal bone. 1: The mastoid emissary veins. 20 Figs. 2: The squamous and petrosquamous emissary veins. 9 Figs.

PROBY, H.: Syndrome vasomoteur du labyrinthe. *Revue d'oto-neuro-ophthal.*, 11: 182, 1933.

Vasomotor syndrome of the labyrinth: Vasomotor labyrinth disturbances may be due to direct vascular lesions or indirectly to subsequent infection, etc. Report of a case in which labyrinth disturbances suddenly supervened and always recurred after an interval of several days. Improvement on simple vascular treatment. (6)

PRUVOST, M.: La re-éducation electrophonoide des sourds. *Echo med. du Nord*, 37:296, 1933.

The electro-phonoid re-education of the deaf: Eight observations showing that the patient's age is not of primary importance, but rather the degree of deafness. Numerous deaf persons can be greatly benefited by this method. (15)

QUESTIONNAIRE ON OTOSCLEROSIS, issued by the Otosclerosis Committee of the Section of Otology. *Royal Soc. Med., Oxford.* June, 1931. *Lancet*, 2:19, 1931. (13)

RABINOWITSCH, S. G.: Blood viscosity in otosclerosis. *Vestnik Sovet Otol.*, 3:231, 1935.

An increased viscosity of the blood serum was found in all stages of otosclerosis (fifty cases), and represents a typical regular phenomenon, apparently proportional to the gravity of the disease.

RAICK: L'otosclérose. *Arch. med. belges*, 81:525, 1928.

Otosclerosis: Review of contemporaneous knowledge and theories; no conclusions.

RAMADIER, J., CHAUSSE, and BZOWSKI: Contribution à l'étude du traitement des otopathies par l'effluvation de haute fréquence. *Ann. des mal. de l'oreille*, 49:880, 1930.

Contribution to the study of the treatment of ear diseases by high frequency effluve. A series of very variegated ear diseases was treated with favorable results in only three cases; in the nine other cases, results were entirely negative, and in one instance, the disturbances actually seemed to be temporarily aggravated. (15)

RATING, B.: Ein sporadischer Fall von blauen Skleren, Knochenbrüchigkeit und Otosklerose. *Endokrinologie*, 13:339, 1934.

Report of a sporadic case of blue sclerotics, bony fragility and otosclerosis, in a man of forty years. Negative outcome of the tests of the endocrinologically important metabolic functions. (4)

REJTO, S.: Otosklerose. *Orvosi Hetilap*, 78:699, 1934.

Discussion of the pathological anatomy and description of the clinical picture of otosclerosis, a disease hitherto somewhat neglected in Hungary. (15)

REUTER, F.: Erfahrungen mit dem U- Strahler. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 31:397, 1932.

Experiences with the U-radiator: Altogether 35 cases of hardness of hearing (not due to otosclerosis) were treated. Four cases of aural tinnitus could not be modified by the U-radiation. (15)

RICCITELLI, E., and FRANCHINI, Y.: Le facteur embryologique dans la pathogénie de l'otosclérose. *Otorhinolaryngol. internat.*, 17:74, 1932.

The embryological factor in the pathogenesis of otosclerosis: Three factors are held essential, i. e., the embryonic (cartilage remnants); glandular dystrophy; some chronic infection such as syphilis. (2; 15)

RICHTER, H.: Operative Luxation des ankylosierten Stapes bei chronischer Mittelohreiterung mit entzündlichem Granulom und doppelseitiger Otosklerose. *Arch. f. Ohren-, Nasen- u. Kehlkopfhlk.*, 139:181, 1935.

Operative luxation of the ankylotic stapes, in chronic middle ear suppuration with inflammatory granuloma, and bilateral otosclerosis, was followed by postoperative labyrinthogenic meningitis and death. Otosclerotic foci were found in both temporal bones. (14)

RODIN, F. H.: Identical hearing defect in identical twins. *Arch. Otolaryngol.*, 17:179, 1933.

Report of deafness with identical hearing defect diagnosed as otosclerosis, (audiogram), in twin sisters sixteen years of age. (1)

ROGGENKAMP, W.: Ueber einen Versuch der psychischen Beeinflussung organisch Schwerhöriger. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 33:94, 1933.

Attempt at psychic modification of organic hardness of hearing, including otosclerosis. Psychotherapy, suggestion, etc., led to definite improvement in hearing in one of the cases. Report of an illustrative case of otosclerosis. (12)

ROLLIN, H.: Bericht ueber die ersten zwanzig operierten Otosklerosefälle. *Arch. f. Ohren-, Nasen- u. Kehlkopfhlk.*, 138:6, 1934.

Report of the first twenty operated cases of otosclerosis, from the Ear-Nose- and Throat Clinic of the University of Hamburg. The advance of the pathologic process is claimed to have been arrested in 90 per cent of the cases. The ear noises subsided or ceased in a high percentage. (14)

—— Ueber operative Behandlung der Otosklerose mit Duralüftung. *Zentralbl. f. Hals- Nasen- u. Ohrenhlk.*, 21:393, 1933.

Operative treatment of otosclerosis with dura ventilation (exposure of the dura). Report of three cases operated upon by Wittmaack's method nine months previously. No further increase of the deafness of the operated ear could be demonstrated by hearing tests. (14)

ROSENFELD, W.: Ein neuer Weg zur Behandlung der Otosklerose. *Med. Klin.*, (Berlin) 28:77, 1932.

New mode of treatment for otosclerosis: Recommendation of adrenalin. Emphasizes need of caution with dosage. (15)

—— Neue Richtlinien im Kampfe gegen Otosklerose. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 134:24, 1933.

New guiding lines in the fight against otosclerosis: Adrenalin, calcium, codliver oil and vigantol medication. Improvement in hearing and relief of tinnitus in about 50 per cent of cases. Considerable improvement of hearing in incipient cases. (15)

RUF, H.: Untersuchungen ueber centrale Hörstörungen. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 36:240, 1934.

Investigations on central auditory disturbances. Demonstrations of hearing curves in the presence of various brain tumors. (7)

RUSKIN, S. L.: The venous circulation of the petrous bone and its clinical significance. *Ann. of Otol., Rhinol. and Laryngol.*, 42:961, 1933.

The venous pathways of the temporal bone play a leading role in the dissemination of infection from the tympanic cavity and the causation of intracranial complications. 5 Figs.

SAIDMAN, J.: Les progrès de la physiothérapie dans la domaine de l'otorhinolaryngologie. *Ann. d'Otolaryngol.*, 4:472, 1933.

Progress of physiotherapy in the domain of otorhinolaryngology: Recommendation of a combined short wave diathermy, polyradiotherapeutic arc and Roentgen ray therapy in the treatment of otosclerosis and aural tinnitus. (15)

SAMUELSON, E., PECK, A., and LEHMAN, A.: Ears and the man. Phila., *F. R. Da is Co.*, 1926.

Studies in social work for the deafened. Bibliography. (12)

SANCHEZ-RODRIGUEZ, I: La reserva alcalina della sangre en las otospongioses. *Revista Espan. y Amer. de Laringol. Otol. y Rinol.*, 22:67, 1931.

The alkaline reserve of the blood in otosclerosis. The calcium- and phosphorus metabolism is surmised to be disturbed through a deficient function of the endocrine glands, especially the ovaries and the anterior pituitary; and the degree of alkalescence of the blood is assumed by French writers as a pathognomonic symptom. Findings in twenty cases of otosclerosis. (9)

—— Alkali reserve in otosclerosis. *Revista Espan. y Amer. de Laring. Otol. y Rinol.*, 24:295, 1933.

The three responsible factors for the development of otosclerosis are local, constitutional and determining. The alkali reserve of the blood is diminished in otosclerosis, but normal in other types of chronic deafness. This alteration of the acid-base balance is of importance for the differential diagnosis.

SCHAEFER, H.: Erfahrungen mit konservativer Diathermie in der Otorhinolaryngologie. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 31:499, 1932.

Experiences with conservative diathermy in otorhinolaryngology and boundary domains. In otosclerosis, no absolute, but relative results were noted. The effective mechanism of the diathermy manifests itself especially in the hyperemic influences inducing a better blood supply of the middle and internal ear, possibly also an improved mobilization of the chain of auditory ossicles. (15)

—— Diathermie in der Behandlung der Hals-, Nasen- u. Ohrenkrankheiten. *Folia Otolaryngol.* Pt. 1, Orig., 22:218, 1932.

Diathermy in the treatment of diseases of the throat, nose and ear. The author employs diathermy in otosclerosis.

—— Die Therapie mit konservativer Diathermie in der Otolaryngologie und einigen Grenzgebieten. *Monatsschr. f. Ohrenhlk.*, 69:833, 1935.

Therapy with conservative diathermy in otolaryngology and some boundary domains: Eleven cases, including nine women. Conservative diathermy treatment can be extensively supported by internal medications. (15)

SCHKLJAR, D. A.: Time of onset of otosclerosis. *Vestnik Sovet Otol.*, 3:241, 1935.

Russian contribution based on a material of 373 cases, the majority belonging to the age group of 31-40 years (79 per cent). Clinical otosclerosis was shown to begin at the age of 21-30 years in 51 per cent of the patients. This diagnosis should be made very cautiously in individuals after the age of fifty.

SCHMIDT, E.: Erblichkeit und Gravidität bei der Otosklerose. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 136:188, 1933.

Heredity and pregnancy in otosclerosis: A study of the family history in 106 cases of otosclerosis; presentation of three new family charts; effect of pregnancy. Otosclerotic heredity does not follow any definite Mendelian law. (1)

SCHNEIDER, R.: Schwerhörigkeit im Bilde der Osteopsathyrose. *Zeitschr. f. Laryngol. Rhinol. Otol.*, 24:362, 1933.

Demonstration on the basis of seven cases seen in the course of one year, showing that the trias of osteopsathyrosis, blue sclerotics and otosclerosis is not a very rare condition. One Figure. (4)

SCHWARZ, M.: Vererbbare Erkrankungen des Ohres. *Jahreskurse f. aerztliche Fortbildung*, 24:52, 1933.

"Inheritable" affections of the ear, including otosclerosis. Cautions against over-valuation of heredity in ear diseases. (1)

SHAMBAUGH, G. E.: The problems of deafness. *Arch. Otolaryngol.*, 14:36, 1931.

Includes a discussion of otosclerosis.

SHAMBAUGH, G. E., and Others: A clinical study of severe deafness in adults. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, 39:103, 1933.

—— Severe deafness in adults. *Arch. Otolaryngol.*, 18:430, 1933.

A clinical study of 65 cases of deafness in adults severe enough to constitute a handicap as due to otosclerosis with stapes ankylosis in the majority of the cases. (70 per cent).

SHAMBAUGH, G. E., JR.: Progressive deafness occurring in identical twins. *Arch. Otolaryngol.*, 17:171, 1933.

Report of two instances of deafness of the same type in identical twins; diagnosed as otosclerosis without stapes fixation; indicative of the importance of heredity as an etiological factor in otosclerosis. (1)

—— Otosclerosis in identical twins. *Journ. Amer. Med. Assoc.*, 104:1216, 1935.

Otosclerosis in identical twins offers a unique opportunity to study the etiology of this common disease. This comparatively rare occurrence has been observed a total of five times. (1)

SIEMS: L'épreuve calorique dans le diagnostic de l'angiospasme cochléaire. *Otorhinolaryngol. internat.*, 14:438, 1930.

The author's caloric test, applied with a hot spurt of oxygen on the

promontory, permits the discovery of the angiospastic character of cochlear involvement, as well as the localization of the seat of the vascular spasm. (13)

SILVEIRA, R.: Otosclerosis. *Revista de Med. y Cir. de la Habana*, 38:58, 1933.

Summary: Clinical forms, diagnosis, therapy.

SIMEONI, C.: Sulla etiopatiogenesi dell'otosclerosi. *Arch. Ital. di Otol.*, 40:378; 413; 481, 1929.

The etiopathogenesis of otosclerosis. Discussion of the etiology and pathology of otosclerosis. Italian text. (8; 11)

SKRIPT, A.: Ueber die Psychotherapie von Ohrenerkankungen. *Monatsschr. f. Ohrenhlk.*, 69:1248, 1935.

Psychotherapy of ear diseases: in the prophylaxis and treatment of ear diseases, psychic and curative factors must be associated. The deafness in otosclerosis is of a combined, organic and psychic character. Here the disturbances of the ordinary mode of life and interruption of the customary activity lead to aggravation of the subjective general condition, with increase of the aural tinnitus, despression of moral energy and loss of faith in the efficiency of the utilized remedies.

SONNTAG, K.: Die prinzipiellen Grundlagen einer Hörverbesserung unter Ausnutzung der Knochenleitung. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 27:430, 1930.

The fundamental bases for improved hearing by utilization of bone conduction: The plan is based on the idea of utilizing, in middle ear deafness, the good bone conduction for sound transmission and replacing by it the air conduction. (13; 15)

SOURDILLE, M.: The surgical treatment of otosclerosis. *J. Laryngol. and Otol.*, 45:601, 1930.

English rendering of French originals. (14)

—— Sur le traitement chirurgical de l'otosclérose. *Revue de laryngol.*, 51:341, 1930.

Surgical treatment of otosclerosis. French text. (14)

—— Résultats primitifs et secondaires de quatorze cas de surdité par otospongiose opérés. *Revue de laryngol., otol et de rhinol.*, 51:595, 1930.

Primary and secondary results in fourteen operated cases of deafness due to otosclerosis. The best functional results were obtained through operation in two stages, with preservation of the ancus applied to the labyrinth wall. (14)

—— Trépanation acoustique du labyrinthe en un temps. *J. belge d'otorhinolaryngol.*, 296, 1930.

Acoustic trephining of the labyrinth in one stage: Window in semicircular canal is covered by a skin flap from the bony auditory meatus. (14)

—— Nouvelles techniques chirurgicales pour le traitement des surdités de conduction. *Ann. des mal. de l'oreille*, 49:10, 1930.

New surgical techniques for the treatment of conduction deafness. French text. (14)

—— Nouvelles techniques opératoires pour le traitement des surdités chroniques progressives ou otosclérose. *Arch. internat. de laryngol.*, 9:200, 1930.

New operative techniques for the treatment of chronic progressive deafness or otosclerosis. Attico-tympanotomy and extra-tympanic trephining of the labyrinth constitute general methods of exploration, control and treatment, the range of which already exceeds the domain of otosclerosis. 9 Figs. (14)

—— Nouvelles techniques opératoires pour le traitement des surdités chroniques progressives ou otosclérose. *Acta Otolaryngol.*, 15:13, 1931.

—— Sur le traitement chirurgical de l'otospongiose. Principes généraux, résultats et méchanismes. Indications opératoires. *Revue de laryngol.*, 53:281, 1932.

The surgical treatment of otosclerosis: General principles; results and mechanisms. Indications for operation. Description of an operation first performed in 1927, based on the improvement in hearing in otosclerosis after opening the semicircular canals system. 8 Figs. (14)

—— Sur le traitement chirurgical de l'otospongiose. *Communication, 2 Congrès International d'otorhinolaryngol., Madrid*, 1932. p. 130.

Surgical treatment of otosclerosis: Summary of the principal results obtained and some deductions based thereon. A period of two years after the last intervention is necessary to judge the definite result. (14)

SPIEGEL, E. A.: Physiopathology of the voluntary and reflex innervation of ocular movements. *Arch. Ophthalmol.*, 8:738, 1932.

The movements of the eyes produced by voluntary or reflex impulses (mainly labyrinthine and retinal, but also partly cochlear impulses) are as a rule associated movements, both eyes moving in the same direction. (6)

SPRUNT, T.: Endocrine features of interest to the otolaryngologist. *Arch. Otolaryngol.*, 11:63, 1930.

The probable explanation of otosclerosis lies in a congenital inferiority of the auditory apparatus, frequently associated with a congenital inferiority of the endocrine system. There is no necessity or justification for assigning to the endocrinopathy an etiologic role in the development of the otosclerosis. (8)

STEIN, C.: Die konstitutionell bedingten Erkrankungen des Ohres. *Wiener klin. Wchnschr.*, 43:461, 1930.

Ear affections due to constitutional causes and their treatment: A distinction can be made between two groups of constitutional anomaly of the auditory organ: congenital, demonstrable at birth; and those appearing in the course of life. (9)

STEINBERG, L.: Ueber die Verteilung und Lage der knorpelhaltigen Interglobularräume in der menschlichen Labyrinthkapsel. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 135:187, 1933.

Distribution and situation of the cartilage-containing marrow spaces in the human labyrinth capsule: Based on the study of forty specimens; found especially in certain portions of the labyrinth capsule but showing no regular arrangement. (2)

STEURER, O.: Das Roentgenbild, ein Hilfsmittel zur Diagnose der Otosklerose? *Passow-Schäfer's Beiträge*, 31:1, 1933.

The Roentgen picture as an auxiliary in the diagnosis of otosclerosis? The roentgenological examination of patients with otosclerosis failed to reveal the otosclerotic focus. The demonstration of good pneumatization of the mastoid excludes adhesive processes as a cause of the deafness, permitting an indirect diagnosis of otosclerosis. (13)

STEVENSON, G. H., and CUTHBERTSON, D. P.: Blue sclerotics and associated defects. *Lancet*, 2:782, 1931.

Deafness was part of the syndrome in one out of four families studied.

TAIT, JOHN: Is all hearing cochlear? *Ann. of Otol., Rhinol. and Laryngol.*, 41: 681, 1932.

Study of the otolith organ. The saccular macula in the air-breathing

vertebrates is presumably a proprioceptor involved in the emission and regulation of voice.

TATO, J. M.: Sobre dos casos de operacion de Sourdille. *Revista de la Asociacion Medica Argentina,* 49:96, 1935 (47:93, 1933).

Two cases of Sourdille's operation (Society Report). One case concerned a young woman with otosclerosis on whom the first step of the operation had been performed in 1933; the second step was delayed, waiting for the necessary instruments; the third step followed two months after the second, strictly according to Sourdille's technic. The results five months later were unsatisfactory. The second patient, a girl of 23 years, with otosclerosis of six years' standing, was operated upon strictly according to Sourdille's three steps procedure in June and November, 1934, and February, 1935. The hearing was considerably improved at the time of the report, in April, 1935. (14)

TANTURRI, V.: Knochenbrüchigkeit, blaue Skleren und Ohrstörungen. *Folia Otolaryngol.,* Pt. Orig., 21:115, 1931.

Bony fragility, blue sclerotics and aural disturbances: In every case of osteopsathyrosis, a careful examination of the ear should be carried out, because latent disturbances of the cochlear apparatus and especially of the vestibular apparatus can be demonstrated. (4)

—— Nueva contribucion al sindrome: fragilidad ossea, escleroticas azules y trastornos auriculares. *Revista Espan. y. Amer. de Laringol.,* 23:97, 1932.

New contribution to the syndrome of bony fragility, blue sclerotics and auditory disturbances: The syndrome is considered as related to abnormal secretions and a disturbance of the biochemistry of the organism. (4)

TERRACOL: Un nouvel appareil pour l'anésthésie continue et à distance. *Ann. d'otolaryngol.,* 1:362, 1933.

A new apparatus for continuous and remote anesthesia. (15)

THIELEMANN: Die Roentgendiagnostik in der Ohrenheilkunde. *Med. Welt.,* 4:76, 1930.

Roentgen diagnosis in otology: This brief sketch does not pretend to cover exhaustively the domain of radio-diagnosis in the specialty, but foresees that the method will soon be in general use also in the difficult domain of otology. 4 Figures. (13)

THIENPONT: Au sujet du diagnostic differential de l'otospongiose. *Ann. d'otolaryngol.,* 4:473, 1935.

Differential diagnosis of otosclerosis: direct radio-diagnosis does not as yet give constant or reliable results. The ear disease may develop in the presence of any degree of pneumatization. (13)

THORNBURG, H. D.: Physiotherapy in the treatment of hearing. *Amer. J. Phys. Therapy,* 7:508, 1931.

Otosclerosis cannot be helped by this type of treatment; as shown by the case of a man of 55 years, with otosclerosis, whose condition was unchanged after one month, when treatment was discontinued. (15)

TILT, E. J.: Elements of health and principles of feminine hygiene. (*Book, Philadelphia, 1853.*) (8)

TINKLE, W. J.: Deafness as a eugenical problem. *Journ. of Heredity,* 24:13, 1933.

The heredity of otosclerosis is explained on the basis of a single pair of recessive genes. Conclusion based on study of the family history in thirty-eight cases of otosclerosis and a review of the literature. (1)

TOBECK, A.: Ueber die Beziehungen zwischen Ostitis fibrosa und Stoffwechselveränderungen. *Zentralbl. f. Hals- Nasen- u. Ohrenhlk.*, 21:395, 1933.

Relations between ostitis fibrosa and metabolic changes. In the presence of a marked destruction of bone, a definite increase in blood calcium is to be anticipated. Where there is new formation of compact sclerotic bone, as in otosclerosis, a diminution in blood calcium is more probable. (4; 9)

—— Die experimentellle Osteodystrophia fibrosa durch Vigantol- Ueberdosierung und ihre Bedeutung für die Otosklerose. *Zeitschr. f. Hals-, Nasen- u. Ohrenhlk.*, 34:188, 1933.

Experimental osteodystrophia produced by over-dosage with vigantol (viosterol) and its significance for otosclerosis: By means of excessive doses of vigantol, fibrous osteodystrophy of the skeletal system and also of the labyrinth bone can easily be produced. The resulting changes are of dystrophic character and are not typical of otosclerosis. 6 Figs. (4; 15)

TORRINI, G., Jr.: Contributo alle conoscenze sulla etiologia dell'otosclerosis. *Valsalva*, 10:347, 1934.

Contribution to the knowledge of the etiology of otosclerosis: Experimental investigations through partial or total removal of the gonads male or female, in young guinea pigs. Examination of serial sections of the petrous bone capsule showed a complete intactness of the labyrinth capsule. A general dysendocrinia or a pluriglandular complex is presumably responsible for otosclerosis. (11)

TURNER, W. G., and MCLELLAN, N. W.: Five cases of osteogenesis imperfecta, in one family. *Canadian Med. Assoc. J.*, 26:659, 1932.

"This small group is of interest because the cases are all in children of the same family; because of the marked degree of the deformity in two cases; and because of a rather peculiar family history." The patients were a boy of six years and three girls of two to six years. The object of this paper is merely to put on record four cases of osteogenesis imperfecta in the same generation of the same family, all having blue sclerae and normal hearing.

VERMES, E.: Zwei Fälle von blauen Skleren, Otosklerose und Knochenbrüchigkeit. *Monatsschr. f. Ohrenhlk.*, 66:495, 1932.

Two cases of blue sclerotics, otosclerosis and bony fragility. Presentation before the Austrian Otological Society, December, 1931. Father of 38 years, daughter of 12 years: The relatively benign course of the auditory disturbance in the father (stationary hearing capacity since 25 years) permits a better prognosis in the child than is generally possible in the presence of otosclerosis.

VIGUERIE, M.: Du traitement médical de l'otospongiose. *J. de méd. et chir. prat.*, 103:885, 1933.

Medical treatment of otosclerosis: Discussion of various methods of medical treatment in use by French otologists. (15)

VILLAR-SANCHO, R., and ALONSO FERRER, E.: Nuestra casuistica de otospongiosi. *Communication, 2 Congrès International d'Otorhinolaryngol., Madrid*, 1932. 140.

Author's otosclerosis material: Contribution based upon 830 cases of deafness, only 36 of which were diagnosed as otosclerosis in its three varieties: stapedio-ankylotic, cochlear and mixed. They consider otosclerosis as a congenital and hereditary dystrophy.

VOSS, O.: Ultraschallwellen im Dienste der Behandlung chronischer Schwerhörigkeit. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 135:258, 1933.

Ultra sound waves in the treatment of chronic hardness of hearing: High frequency sound waves were used in various types of chronic deafness, including otosclerosis, with some benefit in the majority of the cases. (15)

WARWICK, H. L.: Biological factor in analysis of otosclerosis or otitic senility *Laryngoscope*, 41:757, 1931.

Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, 1931. 8 Figures. Bibliography. (1; 9)

WATKYN-THOMAS, and YATES, A. L.: The Principles and Practice of Otology. London, 1932. 555 pp. (13)

WATSON, CHALMERS: On the treatment of deafness of middle ear origin. *Brit. Med. Jrl.*, 1:699, 1902.

Recommendation of bone-marrow (myelocene). (15)

WATSON-WILLIAMS, E.: Otosclerosis treatment with parathyroid extract. *Lancet*, 1:814, 1929. (8; 13; 15)

WEBER, M.: Bone Pathology and its relation to the problem of otosclerosis. *Ann. of Otol., Rhinol. and Laryngol.*, 37:1232, 1928.

—— The bone picture of otosclerosis. *Arch. Otolaryngol.*, 12:608, 1930.

The bone picture of otosclerosis is identical with that of a localized osteodystrophia fibrosa. As biochemical analyses have shown, otosclerosis is always accompanied by a generalized disturbance of metabolism, which might easily have an influence on the general bony system. (2; 4)

—— Zur Theorie der experimentellen Otosklerose. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 26:62, 1930.

Theory of experimental otosclerosis: Bibliography. 10 Figures. 76 references. The bone picture of otosclerosis in its relation to osteodystrophia fibrosa and osteogenesis imperfecta. Postfetal (or fetal) local irritation in typical areas within the labyrinth capsule, and a faulty differentiation of the bone-forming system with left-sided displacement of the bone picture. (4)

—— Osteogenesis imperfecta congenita der Labyrinthkapsel. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 25:345, 1930.

Congenital imperfect bony development of the labyrinth capsule. 11 figures. Bibliography: 38 references. The bone changes of the labyrinth capsule show the common feature of being based on a generalized fetal arrest and faulty differentiation of the bone-forming system, with relative left-sided displacement of the bone picture. (4)

—— Otosclerosis in its histogenic relations to osteodystrophia fibrosa (Ostitis fibrosa). *Arch. Otolaryngol.*, 11:1, 1930.

Nature of otosclerosis as inferred from comparison with osteodystrophia fibrosa. The bone picture of otosclerosis in its relations to osteodystrophia fibrosa and osteogenesis imperfecta. Bibliography: 76 references. 8 Figures. (4)

—— Otosklerose in polarisiertem Lichte. (Otosclerosis in polarized light). *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 28:415, 1930-31.

Otosclerosis represents a separate unit which differs from the bone of the surrounding capsule with regard to the structural elements of its bone: cement substance, cells, fibrils, and at most directly communicates with it by means of the vascular marrow spaces. Otosclerosis is to be interpreted as a new formation after preliminary resorption. (4)

—— Die blauen Mäntel bei Otosklerose. *Communication, 2 Congrès International d'Otorhinolaryngol., Madrid*, 1932. p. 107.

The blue mantles in otosclerosis: A contribution to the pathology of the

labyrinth capsule in man. In 51 examined labyrinth capsules, the bone surrounding the otoclerotic focus showed processes of transformation in the form of perivascular mantles; so-called blue mantles in 28 per cent of the cases. Otosclerosis cannot be regarded generally as a pure primary focal disease, but must be regarded as a disease at least of the whole labyrinth capsule. 12 Figs. (4)

—— Ueber experimentelle Osteodystrophia fibrosa der Labyrinthkapsel beim Hund. *Acta Otolaryngologica*, 17:19, 1932.

Experimental fibrous osteodystrophy of the labyrinth capsule in the dog.

—— Otosklerose und Umbau der Labyrinthkapsel. *Zeitschr. f. Hals- Nasen- u. Ohrenhlk.*, 34:166, 1933.

Otosclerosis and transformation of the labyrinth capsule: The otosclerotic focus is a pathological alteration, a new formation of the bone when old bone has been resorbed; the blue mantles represent the preliminary stage of this process. Brief partial report of a longer article. (4)

—— The blue mantles in otosclerosis. *Ann. of Otol., Rhinol. and Laryngol.*, 42:438, 1933.

A study of the blue, red and mixed mantles surrounding otosclerotic foci in fifty-one specimens, showing the blue mantles consist of pathological plexus-like bone, the red mantles of lamellar bone, the mixed mantles of both types. (4)

—— Otosklerose. *Med. Welt.*, 8:573, 1934.

Outline of the classical picture of otosclerosis and enumeration of early diagnostic difficulties. For prophylactic purposes, marriage or at least procreation should be renounced. (2; 4)

WEBER, G.: Subjektive und objektive Ohrgeräusche. *Folia Otolaryngol.*, 41:1, 1935. (14; 15).

Subjective and objective ear noises are a concomitant phenomenon of two-thirds of all ear affections, the most common of all being vascular bruits. Surgical intervention may enter into consideration in otherwise intractable cases, in the form of: 1) periarterial sympathectomy; 2) reduction of the vascular lumen; 3) ligature of the carotid.

WEILL, S. A.: Quelques considérations sur les épreuves de renforcement. *Otorhinolaryngol. internat.*, 15:99, 1931.

Some considerations on re-enforcement tests: Re-enforcement tests are convenient and easily applied. In his practice, W. utilizes two varieties of re-enforcement, the "forced Weber" and the undulatory re-enforcement. The two tests complete and control one another. (13)

WELLES: The measurement of certain aspects of personality among hard of hearing adults. *Teachers Col.. Contrib. Education*, 1932. (12)

WERNER, C. F.: Die Wachstumsprozesse in der normalen Labyrinthkapsel und ihre Beziehungen zur Otosklerose. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 129:128, 1931.

Processes of growth in the normal labyrinth capsule and their relations to otosclerosis: The growth processes of the labyrinth capsule are connected with only a superficial or partial removal of the enchondral layer, but do not presuppose an internal structural change. The normal histological findings in man and in the hen do not contraindicate, but rather support the stasis theory. (5)

—— Ueber die Wittmaacksche Stauungstheorie der Otosklerose. *Folia Otolaryngologica*, Part 2. (Ref.) 35:141, 1932.

Concerning Wittmaack's stasis theory of otoclerosis. (5)

—— Bau und Entwicklung der Labyrinthkapsel beim Menschen und beim Huhn. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 134:160, 1933.

Structure and development of the labyrinth capsule in man and in the hen: Defence of the use of the hen's capsule as sufficiently like the human, for experimental purposes, as done by Wittmaack for the production of otosclerosis. (5)

WICART, A.: Le traitement de la surdité causée par l'otite moyenne chronique suppurée. *Communication, 2 Congrès International d'Otorhinolaryngol., Madrid*, 1932. p. 151.

Treatment of deafness caused by chronic suppurative otitis media: Mastoid trephining or the radical treatment of chronic otorrhea, which endanger hearing, to be avoided as far as possible, infection and thickening of the mucosa to be guarded against under strict avoidance of all sclerosis-inducing medication, such as alcohol. (15)

—— Le traitement de la surdité causée par l'obstruction tubaire et l'otosclérose consécutive. *Communication, 2 Congrès International d'Otorhinolaryngol., Madrid*, 1932, p. 158.

Treatment of deafness caused by tubal obstruction and subsequent otosclerosis. In middle ear lesions inducing otosclerosis and deafness, phagocyte therapy is recommended, in the form of iodine solution. (15)

WILHOLM, B.: Ueber den feineren Bau der knöchernen Labyrinthkapsel von Hund und Katze. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 136:24, 1933.

The finer structure of the bony labyrinth capsule in dogs and cats: Discussion of the histological structure of the capsule in these animals. The canine labyrinth capsule resembles the human more closely than does the feline. (11)

WILLIGE, H.: Ueber Schwerhörigkeit in der Schwangerschaft. *Arch. f. Ohrenhlk.*, 120:133, 1929.

Hardness of hearing in pregnancy. Otological examination of 35 women without indications of otosclerosis, in the sixth to tenth month of pregnancy, showed in the majority of the cases a strong secretion of cerumen in the auditory meatus, a lipoid arc at the tympanic membrane and corneal margin, as well as a mild internal hardness of hearing. This has nothing in common with otosclerosis.

WILSON, J. GORDON: What America is doing in the study of progressive deafness. *Trans. Amer. Laryngol. Rhinol. and Otol. Soc.*, 38:390, 1932.

Report on the studies of Bast, Crockett, Davenport and Aub, on the ossification of the labyrinth capsule; the heredity of otosclerosis; calcium metabolism in otosclerosis.

—— Review of some of the work being done in America on otosclerosis (Embryonic, metabolic, genetic) 2. *Congrès International d'Otorhinolaryngol., Madrid*, 1932. Section on Otosclerosis, 1:69, 1932.

—— Report of Committee on Progressive Deafness. *Trans. Amer. Otol. Soc.*, 22:45, 1932.

Summary of Bast's findings on the development of the petrous temporal bone; Crockett's findings on the influence of body calcium on otosclerosis; and Davenport's genetical survey of otosclerosis.

WILSON, J. GORDON, and ANSON, B. J.: Form and structure of an area of otitic sclerosis in the temporal bone of an adult. *Arch. Otolaryngol.*, 18:291, 1933.

Macroscopical and microscopical description of an otosclerotic focus in the temporal bone of a male adult, with special regard to the shape of the "sclerotic nodule" and its relation to the immediately adjacent portion of the labyrinth. In sections the diseased bone stained so deeply red with eosin that it was readily distinguishable from the normal bone surrounding it on examination with the naked eye. (4)

WILSON, J. GORDON: Fissula ante fenestram and the adjacent tissue in the human otic capsule. *Acta Otolaryngologica*, 22:382, 1935.

The histology of the area between the foramen ovale and the cochlea is one of special interest to the otologist because it is the site of predilection for otosclerosis. A conception of the extent and diversity of its histological components can be well got by reconstruction from serial sections. It contains the fissula ante fenestram and is one of potential activity even in older individuals. The significance of the fissula is not known. 15 Figs. (4)

—— A new approach to the study of otosclerosis. *Arch. Otolaryngol.*, 21:494, 1935.

Anatomic study based on reconstruction of the area between the oval window and the cochlea, the seat of predilection for otosclerosis. Rather than as a hopeless degenerative process otosclerosis should be interpreted as a congenital weakness which succumbs to environmental factors. (4)

WILSON, J. GORDON, and ANSON, BARRY J.: Histologic changes in the temporal bone in osteitis deformans (Paget's disease). *Arch. Otolaryngol.*, 23:57, 1936.

From the Departments of Otolaryngology and Anatomy, Northwestern University Medical School. This investigation was conducted in 1935 under the auspices of the Central Bureau of Research of the American Otological Society. Observations on the histological features in a case of Paget's disease, in the rather specialized tissue of the petrous portion of the temporal bone. 43 Figs.

WIRTH, E.: Otosklerose und Kriegsdienstbeschädigung. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 126:127, 1930; 128:157, 1931.

Otosclerosis and damage sustained in warfare. In a case of advanced hardness of hearing due to otosclerosis with involvement of the internal ear, in a man of 37 years, a causative connection between military service and otosclerosis did not seem probable and the claim was rejected.

WIRTH, E., and CURTH, H.: Kurvenmässige Darstellungen des Otoskleroseverlaufs. *Arch. f. Ohren- Nasen- u. Kehlkopfhlk.*, 136:202, 1933.

Graphic record in the form of curves showing the course of otosclerosis and confirming the usual data on the course and age of onset of the process.

WITTMAACK, K.: Ueber sogenannte experimentelle Hühnerotosklerose. *Acta Otolaryngologica*, 14:228, 1930.

So-called experimental hen otosclerosis: Demonstration with colored microphotographs, of the results of artificial venous stasis in the hen, on the anatomical structure of the labyrinth bones. Comparison with equally projected specimens of human otosclerosis points to the similarity of microscopical sections obtained in experiments and those of human otosclerosis. (5)

—— Etiology and pathogenesis of otosclerosis. *Arch. Otolaryngol.*, 14:186, 1931.

These deductions show that in all probability the pathogenesis of otosclerosis may be explained by the author's theory propounded in 1919.

This theory is based on noteworthy facts and especially on experiments that may be repeated for verification. (5)

—— Die Ursache der Otosklerose. *Arch. f. Ohren-, Nasen- u. Kehlkopfhlk.*, 129: 150, 1931.

The cause of otosclerosis: A suggestion for causative treatment. Three new cases of otosclerosis show again the bone vein emerging from the bone focus extraordinarily dilated as compared to the behavior of the corresponding vein in the normal labyrinth capsule. A suitable operative intervention, with division of the veins, has been performed on the cadaver, by Mathe, on the author's suggestion. (5)

—— Ueber experimentelle Otosklerose Forschung beim Huhn. *Folia Otolaryngologica*, Pt. 2 (Ref.), 35:100, 1932.

Experimental investigations of otosclerosis in the hen: Abstract of a Danish Society Report presenting a review of his experimentation on hens; findings and conclusions. (5)

—— Betrachtungen zum Otoskleroseproblem. *Acta Otolaryngologica*, 18:217, 1933.

Considerations on the otosclerosis problem: Arguments for the theory of venous stasis as the cause of otosclerosis, based on the normal anatomy of the labyrinth capsule, the peculiar pathological characteristics of the otosclerotic foci and experimentation on hens. 9 Figs. (5)

—— Zur dritten Mayerschen Otosklerose-Hypothese. *Arch. f. Ohren-, Nasen- u. Kehlkopfhlk.*, 134:164, 1933.

Comments on O. Mayer's third otosclerosis hypothesis: Further experiments in support of the venous stasis theory; similarity of experimental otosclerosis in hens and human otosclerosis. 3 Figures. (5)

—— Operative Behandlung der Otosklerose mit Duralüftung. *Klin. Wchnschr.*, 12:658, 1933.

Operative treatment of otosclerosis with dura ventilation. Description of the operative exposure of the dura over the tegmen tympani and division of the venous blood vessels. Performance of the operation in six cases; four times some improvement or arrest of the progress of the disease. (14)

WODAK, E.: Vier Jahre Arsenbehandlung der Schwerhörigkeit. *Monatsschr. f. Ohrenhlk.*, 63:444, 1929.

Four years' arsenic treatment of otosclerosis. By means of arsenic medication, the decay of the auditory capacity in some incipient and moderately grave cases of progressive deafness may be arrested and perhaps the hearing be permanently restored in favorable cases. (15)

WOODS, R.: The treatment of otosclerosis. *J. Laryngol. and Otol.*, 44:372, 1929.

Intra-tympanic injection of liquor iodi B.P. diluted to one-tenth its strength, more stable than the tincture, made not oftener than once a fortnight, gave good results in early cases. (15; 8; 13)

WORMS, G.: Traitement des vertiges et des bourdonnements par les courants electro-anésthésiques. *Revue d'oto-neuro-oculist.*, 8:442, 1930.

Treatment of vertigo and tinnitus by electro-anesthetic currents.

YATES, A. L.: Working hypothesis for research on otosclerosis. *J. Laryngol. and Otol.*, 43:852, 1928.

Formulation of the hypothesis that clinical otosclerosis is at times the terminal stage of subacute otitis media in which the products of the inflammation of the middle ear are conveyed away by the eustachian tube. (13)

YAZUJIAN, D. M.: Diathermy in the treatment of chronic deafness. A new technic. *Laryngoscope*, 45:230, 1935.

Importance is attached to diathermy treatment with employment of two electrodes, the electric current passing from one electrode to the other. "Ototheramy" without vibratory massage and inflation immediately following it is not of much value. Chronic nerve deafness and otosclerosis are not benefited by it.

YEARSLEY, MACLEOD: Anomalous case of deafness successfully treated by the electrophonoid method. *Brit. J. of Child. Dis.*, 25:116, 1928.

Report of a case of deafness of doubtful origin in a boy of ten years. Highly satisfactory results in response to the Zünd-Burguet method of treatment. (15)

—— Problem of otosclerosis. *Practitioner*, 122:348, 1929.

Every case of otosclerosis exhibits some or more of the cardinal signs of familial chronic intestinal intoxication together with a marked degree of mal-coordination. Selection of seven cases, all of whom had typical otosclerosis with paracusis Willisii, and all had chronic familial intestinal intoxication. All received treatment of the general condition. (7)

—— Intestinal toxemic deafness in children. *Brit. J. Child. Dis.*, 26:116, 1929.

The form of deafness known as otosclerosis, which has puzzled otologists for two generations, is primarily a toxemia affecting the cortical cells of the auditory area, the change in the labyrinth capsule being in all probability trophic in nature. (7)

—— A theory of otosclerosis. *Congrès. International d'Otorhinolaryngologie*, Compt. rendus, 1:437, 1929. Congress held in 1928; Report published latter part of 1929.

—— Further observations on the electrophonoid treatment of deafness. *Practitioner*, 128:631, 1932.

This treatment is a valuable method in a large number of cases of deafness, and its employment is founded upon a strictly rational basis. It is this physiological foundation that makes it successful in otosclerosis. (15)

—— Otosclerosis: its etiology and treatment. *Practitioner*, 131:606, 1933.

The primary change in the brain is probably the true starting point of otosclerosis, and this points to the direction which future pathological research should take. The hypothesis of the intestinal origin of otosclerosis has a very practical and important bearing upon treatment. (7)

—— Otosclerosis, with special reference to etiology and treatment. *Monograph*, London, 1933. (7)

—— A case of deafness due to rickets. *Brit. J. Child. Dis.*, 30:194, 1934.

"This case is of great interest for two reasons· the rarity of rickets as a distinct cause of deafness in children, and the suggestion it conveys of a connection between rickets and otosclerosis." The patient, a completely deaf girl of four and a half years, was admitted to the hospital with a history of deafness from rickets when twelve months old. (4)

ZIMMERMANN, A.: Ohrensausen und seine Behandlung. *Fortschr. d. Therapie*, 6:225, 1930.

Aural tinnitus and its treatment: Otosclerosis is often associated with subjective ear noises, which may be the result of a vaso-constrictor angioneurosis. The treatment must, if possible, be adapted to the cause of the tinnitus. (6; 15)

Clinical Notes.

XXXIV.

OTITIC HYDROCEPHALUS WITH A SUGGESTION AS TO ETIOLOGY.*

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Otitic hydrocephalus, so named by Symonds,[1] is a state of intracranial hypertension, associated with an increased pressure or an increased amount of cerebrospinal fluid, occurring during otitis media or mastoiditis. This entity includes those conditions that were formerly known as pseudomeningitis, pseudobrain abscess, serous meningitis, aseptic meningitis, ependymitis serosa, meningeal hydrops, etc. Symonds suggested the term of otitic hydrocephalus, "a title which implies no acute process of inflammation, and being deprived of the qualification internal or external, will include fluid both within the ventricles and within the subarachnoid space."

The symptoms of this condition are those of increased intracranial pressure, headache, drowsiness, photophobia, choking of the optic disc, increase in the amount or pressure of the cerebrospinal fluid, nuchal rigidity, vomiting, positive Kernig's sign, and at times trigeminal pain, a sixth nerve palsy or other focal symptoms.

Otitic hydrocephalus has been most clearly described by Symonds, and a review of the literature is included in his original article, to which the reader is referred. Mere repetition would be superfluous. This condition usually occurs in childhood or adolescence. The etiology is obscure. Some authorities suggest that it may be due to an excessive secretion from the choroid plexus or a defective absorption through the arachnoid villi.

In a previous publication we[2] suggested that venous stasis of the intracranial circulation may prevent absorption of the cerebro-

*From the Department of Otology, Temple University, School of Medicine.
Presented before the Philadelphia Laryngological Society, December 3, 1934.

spinal fluid. This stasis may be produced by disease either in the mastoid process or other parts of the temporal bone, or by anomalies of the cranial veins. Roentgenologically, some of these cases presented variations in the size of the lateral sinuses and jugular foramina.

Leidler, quoted by Symonds, reported a case in which ligation of the internal jugular vein for infection of the lateral sinus resulted in severe headache and papilledema, lasting for several weeks, with ultimate recovery. He suggested that the state of increased intracranial pressure was due to obstruction of the venous outflow.

Leshin[3] quotes a similar case. "Case 3. S. W.—Following ligation of the jugular, the patient remained unconscious for several hours, with a rapid feeble pulse, dilated pupils, a Babinski sign on the left, ankle clonus, the Kernig phenomena and considerable restlessness. The clinical picture was that of shock, with some intracranial pathology." Leshin commented: "This was a case of otogenic sepsis with no definite evidence of thrombosis of the lateral sinus, complicated by shock and by what appeared to be cerebral edema following ligation of the right internal jugular vein and obliteration of the right lateral sinus."

From our own experience we have two cases that will illustrate the intracranial disturbance that may result from interference with the venous drainage of the cranial vault. Both these cases involved obliteration of the lateral sinus due to septic processes in this vessel.

REPORT OF CASES.

Case 1.—B. L., aged six. Four years ago this patient developed thrombosis of the right lateral sinus following an acute mastoiditis. The right lateral sinus was obliterated. The left lateral sinus in this patient was much smaller than that of the right. The patient made an uneventful recovery from the sinus thrombosis. As an aftermath of this illness, with each of his frequent episodes of acute right otitis media he manifested signs of intracranial disturbance such as hemicrania, drowsiness, nuchal rigidity and choking of the optic disc on the right. These symptoms were relieved by myringotomy or when spontaneous perforation of the ear drum occurred. In addition, the use of magnesium sulphate, glucose intravenously, fluid limitations, and even lumbar puncture were necessary at times to relieve the intracranial pressure.

Case 2.—M. W., age three. Following obliteration of the right lateral sinus for lateral sinus phlebitis, this patient developed a sixth

nerve palsy and choking of the optic disc on the right side. In addition, headache, vomiting and nuchal rigidity. The acute symptoms disappeared following the relief of increased intracranial pressure by the use of magnesium sulphate, glucose intravenously and lumbar puncture. The sixth nerve palsy persisted for several months.

This case is somewhat similar to Case 2, page 63, described by Symonds. His patient was subjected to exploratory puncture of the brain on the supposition that a brain abscess was present. No pus was found, merely an increased intraventricular pressure being noted. In the discussion of this case he admits that a preliminary spinal puncture would have established the diagnosis and saved the patient the unnecessary brain surgery.

We have previously shown, and it has been stated by others, that it is not necessary to ligate the jugular vein to produce venous obstruction. Disease of various portions of the temporal bone will produce venous stasis and obstruct venous outflow. If the temporal bone pathology is on the side of the larger venous structure (often on the right), then the opposite side (if smaller or anomalous) is inadequate to handle the entire burden thrown on it. Thus the state of increased intracranial pressure, or otitic hydrocephalus, may be produced and result in meningeal symptoms.

The diagnostic finding in this condition is an increase in the spinal fluid pressure and in the quantity of spinal fluid. The cellular and serologic elements of the spinal fluid may or may not be disturbed. The change in the spinal fluid cell count depends on the type of case seen.

There are several classes: (1) In the acute cases, which are seen early and which manifest early signs of increased intracranial pressure, a spinal puncture will show the fluid to be clear and under increased pressure. These cases are promptly relieved by the puncture. The irritative focus in the mastoid must, of course, be removed in order to prevent a recurrence of the symptoms.

(2) In the type of case of longer duration which manifests meningeal signs, examination of the spinal fluid may show increased pressure and an increase in the number of cells. Should no organisms be found on direct smear or culture, this increase of cells should not be taken as necessarily indicative of a beginning suppurative meningitis, but rather as a reactive state of the meninges. Dwyer[1] explains this as follows: "A cloudy spinal fluid without organism does not necessarily mean generalized meningitis. The cloudiness may be due to an effusion of the cells into the spinal fluid, the result of a tissue

reaction due to organisms on the outside of the dura. There is an exact analogy in nature, well known to the ophthalmologist. In ulcer of the cornea the anterior chamber of the eye is often full of pus (hypopyon). The hypopyon is usually sterile, and the offending organism, the pneumococcus or streptococcus, is on the outer surface of the cornea; the tissue reaction is evidenced by sterile pus (effusion of cells) in the anterior chamber."

Another important factor in otitic hydrocephalus that aids in the establishment of the diagnosis is that the same spinal drainage that gives us the clue to the condition also acts as the therapeutic measure which effects the cure. In many cases the single puncture will produce an amelioration of all symptoms.

CASE 3.—L. F., male, white, age seven. The patient was admitted to the Otology Service of the Temple University Hospital on May 17, 1930. The chief complaint on admission was pain in the right ear, right temporal headache and discharge from the right ear for the past three weeks. Three weeks prior to admission a right myringotomy had been performed; the pain was relieved and it was followed by a discharge of thick creamy pus. This continued for one week, and when the drainage ceased the myringotomy was repeated. Following the examination on the day of admission, the patient was referred to this hospital with a diagnosis of acute right mastoiditis.

The past medical history revealed that the patient has had chickenpox one year ago, which was followed by a right otitis media, from which he recovered. The child also has had measles, mumps and whooping cough.

The physical examination revealed a thin, anemic, emaciated child, with a temperature of 100.4°, pulse 112, respiration 32. The examination was essentially normal except for the right ear, which showed an acute suppurative otitis media with acute mastoiditis. A right simple mastoidectomy was done on the day of admission. A hemorrhagic type of mastoid, with softened and necrotic bone, was found at operation. The patient did well postoperatively and was discharged on May 25, 1930, eight days after admission.

On June 8, 1930, thirteen days after his discharge from the hospital, he developed nausea, vomiting and photophobia. There was a temperature of 101°.

Examination at this time revealed a thin, emaciated child, showing evidence of dehydration. There was marked photophobia. The conjunctivæ were paler than normal and a slight ptosis of the right

upper lid was present. The left ear was normal. The right ear showed a postauricular scar, which was open at the lower angle and from which drained thick, creamy pus. There was a right peripheral facial palsy present, nystagmus to the left, and a slight disturbance in cöordinated movements of the right upper limb with increased reflexes on that side.

The eye examination revealed the following: Vision, O. D., 20/50; O. S., 20/40. There was a slight paresis of the right upper lid; the right palpebral fissure was smaller than the left. In the right eye the cornea was clear, the conjunctiva slightly congested, the anterior chamber normal. The pupils were five millimeters in diameter and reacted to light and accommodation. Muscles were normal and convergence was full. Nystagmoid movements were present in the fields of rotation. The fundus of the right eye showed the media clear, the disc oval, and hazy as to outline. On the nasal side there was a circumscribed ring of pallor around the disc which itself was pale. The vessels were overful, the veins especially were congested The macula and periphery were negative. The left eye was normal.

The following opinion was expressed by Dr. Winkelman of the Neurologic Department: "The history of the vomiting coming on suddenly, the pain over the eye and the course of the case would suggest a brain abscess. I would advise inactivity at present as regards the brain lesion, until symptoms are more definite and localization and walling off have occurred."

The physical examination by Drs. Schless and Pressman was negative, except for the findings detailed above.

On June 15, 1930, two days after readmission, the child seemed brighter; however, neurologic signs were still present.

On June 16, 1930, seven days after his readmission, the child complained of severe pain over the eyes and in the right ear. He seemed drowsy and the nystagmus and the seventh nerve palsy continued. The eye examination showed no change.

June 18, 1930, nine days after readmission, the patient vomited twice and was dull and apathetic. The nystagmus was more marked to the left. The weakness of the seventh nerve remained unchanged. Definite nuchal rigidity continued and there was a marked cerebellar incoordination to the left upper limb. Examination revealed abortive ankle clonus bilaterally but no Babinski or Kernig. The eye examination on this date showed the right disc as being more hyperemic than it was on the previous examination. There were two diopters of chok-

ing. The above findings were suggestive of an exacerbation of the patient's condition and a lumbar puncture was suggested. Lumbar puncture done on this date, in the prone position, revealed a pressure of 12 millimeters of mercury with cloudy spinal fluid. A study of the fluid revealed 555 leucocytes per cubic millimeter, a sugar of 50 milligrams per 100 cubic centimeters and a trace of globulin. No organisms were present on smear or culture; 30 cubic centimeters of fluid were removed.

June 19, 1930, ten days after admission; Nuchal rigidity was still present, but it seemed less marked. There was still a marked nystagmus on looking to the left. The facial paralysis was improving. The left upper limb still showed a definite coarse tremor. The finger to nose test showed the presence of dysdiadockokinesis. From the therapeutic response to the lumbar puncture, the suggestive diagnosis of serous meningitis as the result of a neighboring inflammatory lesion was made. A Bárány test was done by Dr. Winston; this suggested a posterior fossa lesion, probably extradural. The spinal drainage was repeated on this date.

June 20, 1930, eleven days after admission: The mastoid wound was reopened and the granulation tissue was removed. Necrosis of the remaining cells in the zygomatic region was found, and along the knee of the lateral sinus and in the region of Trautman's triangle pathology was noted. A right radical mastoidectomy was performed; exposure of the cerebral and cerebellar dura and the lateral sinus were found to be normal when inspected.

June 21, 1930, the day following the second operation: The temperature dropped to 99.4°. The child did not complain of headache, nausea, vomiting or vertigo. He appeared bright and cheerful, and was not so drowsy. His cerebration was alert; there was no demonstrable nuchal rigidity, no Kernig, Brudzinski, Babinski or ankle clonus. The finger to nose test was much improved; there was no nystgamus or facial palsy. An improvement in the condition of the eye grounds was also noted. A spinal puncture done on this date revealed the fluid to be under normal pressure and with a normal cell count. From this time on, recovery was uneventful and the patient was discharged on July 6, 1930, twenty-seven days after his second admission.

A summary of the salient features of this case is as follows:

1. Case admitted to the hospital May 17, 1930.

2. A history of a discharging ear for three weeks previous to admission. The ear drum was incised twice while at home.

3. Acute mastoiditis, right, with mastoidectomy; discharged from the hospital in eight days.

4. Thirteen days after being sent home from the hospital an exacerbation of symptoms, nausea, vomiting, photophobia, with temperature 101°, right peripheral facial palsy and nystagmus.

5. Thick pus from the middle ear and mastoid.

6. Possibility of a brain abscess, petrositis, or serous meningitis considered.

7. Lumbar puncture done with count of 555 leucocytes and pressure of 12 mm. Hg. Spinal puncture was performed on three occasions.

8. Ten days after the second admission the mastoid wound was reopened, granulation tissue curetted, necrosis of mastoid and zygomatic region found. Right radical mastoidectomy: exposure of the lateral sinus, cerebral and cerebellar dura, all of which appeared normal.

9. Improvement of symptoms and patient discharged as cured, twenty-seven days after second operation.

This case presents a problem similar to the one described by Symonds[1] (case No. 3, page 65), in which there was a question of a focal brain lesion. This type of patient has often been subjected to unnecessary intracranial surgery with exploratory puncture for brain abscess, and on failure to locate pus was termed pseudo-brain abscess. We feel that the therapeutic response to the spinal drainages and the removal of the primary focus places it in the category of otitic hydrocephalus.

It might be added that since this case occurred in 1930, before the appearance of Symonds' description, it was classified as serous meningitis.

CASE 4.—E. G., female, white, age 10. This patient was admitted to the Temple University Hospital, Otology service, on January 28, 1931, with compliant of pain in the left ear, worse at night, and pain and swelling in the left mastoid region. This illness began on January 20, 1931, eight days previously. The diagnosis on admission was a left acute recurrent mastoiditis.

The past medical history revealed that in May of 1930, the patient had scarlet fever, which was complicated by a left mastoiditis, and mastoidectomy was done at the Philadelphia Municipal Hospital.

The patient also had measles and pertusis. A tonsillectomy and adenoidectomy had been done in November of 1930.

The physical examination was negative except for the left acute otitis media and mastoiditis, and an anterior cervical adenopathy on the left side. At operation, performed on January 29, 1931, the findings were chronic suppurative otitis media, localized meningitis, and localized lateral sinus phlebitis. The operation was a left radical mastoidectomy, with a decompression of the middle fossa and the lateral sinus. The gross findings noted at operation were: the periosteum over the mastoid was very thick and adherent; pus, necrotic cells and granulations were present in the mastoid cavity, and granulation tissue and cholesteatomata were found invading the middle fossa. Following this operation the patient's condition remained good and she was discharged on February 10, 1931, thirteen days after admission.

The child seemed to be doing well following the operation until March 8, 1931, twenty-six days after her discharge from the hospital, when she contracted a pharyngitis and an actue choryza. The patient also developed a high fever and complained of frontal headache. She became very restless; nuchal rigidity and general hyperesthesia of the body were noted. The child cried a great deal and vomited several times. In view of the above history and findings she was readmitted to the hospital on March 9, 1931, with a provisional diagnosis of otitic meningitis secondary to mastoiditis or lateral sinus phlebitis.

Positive findings on examination were nuchal rigidity, hyperesthesia, enlargement of the anterior cervical glands, and tenderness in the left knee and leg. Kernig's sign was present. The eye grounds revealed that the veins of both discs were slightly fuller than normal.

The x-ray examination made on admission was reported to us as follows: "The left mastoid has been operated on and shows that most of the air cells have been obliterated or removed. Some air cells still remain in the mesial portion of the left petron, but those do not contain air; they give the x-ray appearance of an exudative mastoiditis. We can make out no evidence of bone necrosis. We are able to say from the present films that the left lateral sinus is unusually large and that it is much larger than the right."

A lumbar puncture was done on the day following admission. The pressure was 16 mm. Hg., 5 cc. of spinal fluid was removed for study. This revealed an increased amount of globulin; the sugar was 76.5 mgm. per 100 cc., the leucocytes were 865 per cubic millimeter.

No organisms were seen on direct smear. Hematology on admission, March 10, 1931, was Hb, 70 per cent; R. B. C., 3.57; W. B. C., 23,150; P. M. N., 70 per cent; L. L., 30 per cent.

It was decided to explore the operative site again. This was done on March 10, 1931. The former incision was reopened, the old granulation tissue was removed, the cells from the region of the jugular bulb were removed and the lateral sinus from the sigmoid down to the bulb was exposed. The cerebral dura was uncovered for an area about the size of a silver dollar. The tegmen was removed close to the superior semicircular canal. The dura was incised and a small quantity of cloudy spinal fluid escaped.

Postoperative course: March 11, 1931, one day after admission, 55 cc. of cloudy spinal fluid with a pressure of 6 mm. Hg. was removed; the cell count at this time was 200 W. B. C. per cubic millimeter. The patient was drowsy; nuchal rigidity and Kernig's sign still present. She was given 50 cc. of 50 per cent glucose intravenously and 20 cc. of antimeningococcus serum intraspinally, as a prophylactic measure.

March 12, 1931, two days after admission: Spinal tap again revealed a pressure of 6 mm. Hg., and 50 cc. of cloudy spinal fluid was removed. The cell count was 129 W. B. C. per cubic millimeter. The day following her temperature, pulse and respiration were normal; the patient was alert and seemed well oriented as to time and place and she partook of liquids freely.

March 14, 1931, five days after operation: The temperature was 100° (rectal), the patient was cooperative, but a slight nuchal rigidity and Kernig were still present. The mastoid wound was draining freely.

March 15, 1931, six days after admission: Spinal tap was repeated. The pressure was 6 mm. Hg.; 60 cc. of fluid was removed and cell count was 213 per cubic millimeters. The patient's general condition seemed much improved.

March 18, 1931, nine days after operation: The patient's general condition remained good. The temperature was normal, and only a questionable Kernig's sign remained. Lumbar puncture revealed a pressure of 6 mm. of Hg. and 40 cc. of spinal fluid were removed.

She was apparently convalescing nicely until March 29, 1931, twenty days after admission, when she developed a lower left facial palsy. No ocular palsy was present. A slight elevation of the temperature was noted.

The mastoid wound healed nicely, the neurologic signs disappeared, except the left facial weakness, and on April 15, 1931, forty days after admission, the patient was sent home, apparently in good condition. There was, however, a slight amount of drainage from the left external auditory canal still present.

July 31, 1931, three months after the discharge from the hospital, the patient again began to have pain, and postauricular swelling and fluctuation on the left side with drainage from the left ear. This wound was reopened and no pus was found. The dura was under increased tension and fluctuated on palpation. An incision was made into the dura; this was followed by a free escape of spinal fluid. There was a slight rise in temperature following the operation. The patient was kept under observation, redressed daily, and she was again sent home on August 29, 1931, twenty-nine days after admission, with the wound healing and a scant discharge from the left ear.

The patient seemed to do well at home, until September 19, 1931, twenty days after the last discharge from the hospital, when she again developed temperature and vomited. There were no chills or convulsions. Examination failed to reveal any abnormal neurologic signs. The eye grounds were normal, there were no visual field defects. A lumbar puncture showed the spinal fluid to be under normal pressure and the cell count was normal. A Tobey-Ayer test gave a normal response to jugular compression. Vestibular examination gave a fairly normal response. The patient was kept under observation for several days and then was again discharged from the hospital. Since that time she has been in fairly good health, with the exception of an occasional discharging left ear.

A summary of this case is as follows:

1. January 28, 1931.—Patient, age 10, female, admitted to the hospital with a diagnosis of left acute suppurative otitis media and acute recurrent mastoiditis of eight days' duration.

2. At operation on February 10, 1931, cholesteatoma was found and a radical mastoidectomy was done. The patient was discharged from the hospital ten days after admission.

3. March 8, 1931, twenty-six days after her discharge from the hospital, she again began with temperature, frontal headache, vomiting, hyperesthesia, nuchal rigidity and Kernig. Patient was readmitted to the hospital with a diagnosis of otitic meningitis.

4. A spinal puncture was done on admission, and it revealed a pressure of 16 mm. Hg. and 865 W. B. C. per cubic millimeter.

5. March 10, 1931, two days after her second admission a secondary operation of the mastoid wound was done with a decompression of the middle fossa; this was followed by an incision of the dura with an escape of cloudy spinal fluid.

6. March 11, 1931, a spinal drainage was done, and although the spinal fluid pressure was only 6 mm. of Hg., still there was an increased amount of spinal fluid—50 to 60 centimeters being removed at each drainage. This was repeated on five separate occasions, and each time an increased amount of fluid and an increased cell count was noted.

7. It is important to note that the x-ray showed an extremely large lateral sinus on the left side and a very small one on the right.

8. April 15, 1931, the patient was discharged—improved—thirty-six days after her admission to the hospital.

9. Exacerbation of symptoms on July 31, 1931, three months after previous discharge with abatement of symptoms.

10. One month following this admission, again an exacerbation of symptoms and again there was an improvement.

CASE 5.—R. W., male, age seven, white, admitted to the Otology Service of Temple University Hospital, on December 9, 1933, with the diagnosis of bilateral acute mastoiditis, and possible otitic meningitis. The temperature on admission was 105° (rectal). The present condition began five days before admission to the hospital following an upper respiratory infection. The patient vomited once on the day previous to admission to the hospital.

Physical examination revealed a young patient who appeared rather toxic, although he was mentally clear. His cheeks were flushed and there was circumoral pallor present. He demonstrated nuchal rigidity and a slight Kernig's sign on the right.

The otoscopic picture was as follows: The right ear drum disclosed congestion in Shrapnell's membrane; the drum was lusterless and slightly full. Hyperesthesia of the skull was present and also definite mastoid tenderness was noted. Eye ground examination, ocular fields and ocular muscular movements were normal.

The x-ray examination revealed the following: The cells of both mastoids are abnormally dense. Practically no air filled mastoid cells were visible on either side. In the region of each auditory canal

one finds the cell structure somewhat irregular, suggesting the possible presence of suppuration with destruction of the cell wall.

The impression of the right lateral sinus is larger than that of the left. The right jugular foramen is likewise much larger than the left, although both of these are within normal limits for size. There are no cells in either petrous pyramid. The right maxillary is completely filled with a homogeneous density. In the left maxillary there is also heavy marginal density suggestive of polypoid thickening.

Diagnosis following the above examination was bilateral acute suppurative mastoiditis, possible otitic meningitis and acute bilateral maxillary sinusitis.

Progress: Immediately following his admisison to the hospital a bilateral myringotomy was done. This was followed by the release of a small amount of serosanguinous material. A spinal puncture was done on admission to the hospital, which revealed an initial pressure of 26 mm. and 15 cc. of fluid. This was removed and found to contain 29 cells per cubic millimeter.

December 18, 1933, the day following his admission, the temperature remained 105° (rectal) and was of the sustained type. Both ear drums were congested and no landmarks were seen. A left central perforation was noted. No drainage followed the myringotomy of yesterday. The Kernig's sign, the nuchal rigidity and the Brudzinski were still present. Both antra were punctured and pus was obtained from each maxillary sinus.

December 8, 1933, one day following admission: The spinal puncture was repeated. The initial spinal pressure was 10 mm. Hg., 12 cc. of fluid was drained; the spinal pressure was six millimeters.

An intravenous injection of Pregl's iodine was given. The blood culture was negative.

December 9, 1933, a bilateral mastoidectomy was performed, with exposure of the dura and the lateral sinus. Pus and necrosis of the cells in both mastoids were found, and an intranasal maxillary opening was performed on the right side. The culture from the mastoid cavity revealed the pneumococcus organisms.

December 10, 1933, the day following operation, the temperature fell to 100° (rectal), and an absence of all meningeal signs was noted. From this time on the temperature remained normal, the mastoid wounds healed by primary union, the convalescence was un-

interruptedly uneventful and the child was sent home in good health December 20, 1933, twelve days after admission.

A summary of this case is as follows:

1. The child was admitted to the hospital with a history of a bilateral otitis media, following an acute upper respiratory infection. This was followed by a sinusitis, bilateral mastoiditis and meningeal symptoms.

2. X-ray revealed a bilateral mastoiditis with a very large right lateral sinus and a smaller left lateral sinus.

3. Spinal fluid pressure was under increased tension with a slight increase of the cell count.

4. Three spinal punctures were done, bilateral mastoidectomy and intranasal maxillary on the right side.

5. Uneventful convalescence ensued and the patient was discharged from the hospital twelve days after admission.

DISCUSSION.

This case may be considered one of meningeal irritation secondary to a suppurative mastoid lesion or may come under the classification of Symond's otitic hydrocephalus, due to the increase of spinal fluid pressure and the increased spinal fluid cell count.

CONCLUSION.

1. X-ray of the lateral sinus and jugular foramina, in addition to pictures of the mastoid process and petrous portion of the temporal bone, are indicated in each case of acute otitis media and mastoiditis. This will acquaint us with the state of affairs in all parts of the temporal bone as well as the state of the venous structures.

2. Symptoms of otitic hydrocephalus are those of early meningeal irritation. The condition may also simulate brain abscess.

3. The diagnosis is made by lumbar puncture and drainage, and by the fact that following this procedure there is usually a disappearance of the alarming intracranial symptoms.

4. Three cases are presented in detail which we believe fall into the category of otitic hydrocephalus, and which recovered in each instance following spinal drainage and removal of the irritative focus of the mastoid.

5. Two cases are presented in abstract. In these patients we believe the appearance of the otitic hydrocephalus followed the oblit-

eration of the lateral sinus. This helps to corroborate our hypothesis that intracranial circulatory disturbances are important factors in the production of this condition.

1915 SPRUCE STREET.

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XXXV.

ADENOCARCINOMA OF THE EXTERNAL AUDITORY CANAL.*

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Cancer of the external auditory canal seems to be a rare lesion, and a search of the literature reveals only a small number of reported cases. There is no doubt that at least some of the cases of aural polypi and granulomata, which were not verified by biopsy, may have been cancer.

Most of the recent papers stress the importance of biopsy in cases of this kind. It is also pointed out that many cases of malignant neoplasm of the middle ear may have had their origin in the external auditory canal.

The feeling that even single case reports would be of help in compiling data is the reason for presenting this paper.

Adenocarcinoma appears to be the least frequent of the malignant neoplasms of the middle ear and external auditory canal.

In a group of six cases of malignant neoplasm of the middle ear Schall[1] reports one case of adenocarcinoma involving the external canal and middle ear. Furstenberg[2] reports a case of adenocarcinoma of the middle ear. There was possible involvment of the external canal in this case, according to the pathologic report. Fraser[3] reports one case with a pathologic report of adenocarcinoma, and a second case, which resembled basal cell carcinoma in some areas and adenocarcinoma in other areas. He called these two cases "malignant sebaceous adenoma."

The above four cases, considering both of Fraser's cases as adenocarcinoma, were the only cases of adenocarcinoma I could find reported in the literature.

Schall's case was alive at time of reporting, with no recurrence, four years and two months after radical mastoidectomy and irradiation. Furstenberg's case was still living four years after operation. Fraser's cases both died, one nine months after operation and the other

*Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Newark, N. J., January 3, 1936.

he reported as slowly dying eleven years after operation. The case I am reporting is still living, without recurrence, seven years and ten months after removal by snare and radio-ionization. This represents a mortality rate of 40 per cent in adenocarcinoma of the middle ear and external auditory canal.

The etiology of adenocarcinoma of the external auditory canal is obscure. Many otologists agree that the irritation of chronic discharge is a predisposing factor. In one case the patient had a purulent discharge for many years. In my case there was no discharge prior to the appearance of the neoplasm, but occurred after the growth had plugged the meatus. History of trauma has been elicited in cases of cancer of the auricle.

REPORT OF A CASE.

CASE 1.—The patient is a married white woman, fifty-five years of age. She was referred to me by Dr. Arthur R. Vaughn, who had made a clinical diagnosis of cancer of the ear.

Her chief complaint was that she had a growth in her right ear which caused earache and a purulent discharge. At times there was slight bleeding.

The family history was negative. The patient has had no chronic diseases. The only severe illness was typhoid fever in early childhood.

Present illness: The patient noticed something growing from her right ear about June 1, 1927. This growth gradually blocked her ear, causing earache, when one month later she consulted a physician, who removed it in his office, apparently without local anesthesia. The doctor, shortly after removal of the growth, went on his vacation and treatment was discontinued.

The growth reappeared shortly after removal and grew quite rapidly for three months, with recurrence of earache and discharge. She was then referred to me, October 13, 1927.

Examination revealed a large red growth protruding from the right external auditory canal and was about 2 x 1½ cm. in diameter. The growth resembled granulation tissue, although it felt more firm in consistency. It was roughly oval in shape and appeared to be attached to the floor of the canal by a narrow base. The mass did not bleed upon manipulation such as granulation tissue does, but a small amount of purulent secretion oozed from around the growth. It was not tender. There was no tenderness over the mastoid nor was there any pain when traction was made on the auricle. There were no palpable lymph nodes.

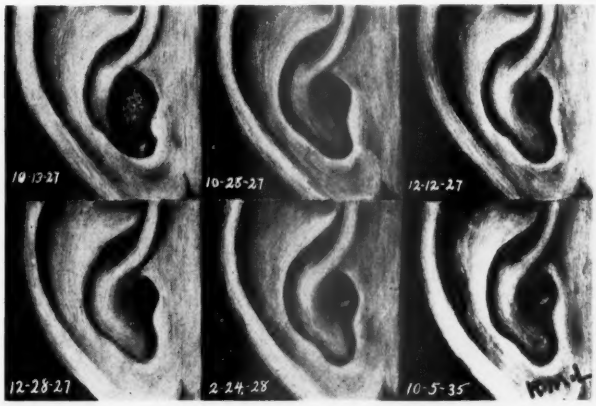

Fig. 1. Oil sketch of ear showing adenocarcinoma of the external auditory canal during and following radio-ionization of uranium sulphate.

10-13-27—Tumor when first seen. 10-28-27—After first section was removed. 12-12-27—After removal of second specimen and radio-ionization treatment. 12-28-27—After radio-ionization treatment. 2-24-28—Shows a scar in the canal after radio-ionization (the case apparently cured). 10-5-35—The scar in the external canal (same appearance as 2-24-28) 7 years and 8 months later.

About one-half of the growth was removed with the snare and sent to the laboratory for histologic diagnosis. The remainder of the neoplasm was treated by radio-ionization at intervals of two or three days. After two weeks of ionization treatment, the growth was much dryer and presented a grayish white top. The discharge was much thinner and pain had decreased greatly.

The greater portion of the remaining growth was then removed by snare and sent to the laboratory with a request to report any changes in the tissue which might have taken place since the first specimen was removed. The report on the second specimen stated that there was no change. The pathologic diagnosis was adenocarcinoma.

Radio-ionization was continued at short intervals. The growth gradually assumed a shrunken appearance and showed a grayish white slough in the center after several treatments. This diminution in size continued until the growth was represented by a small, whitish, elevated point on the floor of the canal near the meatus. This point resembled scar tissue somewhat and seemed to be covered with normal epithelium. Occasionally a small cap of dark cerumen would collect at this point.

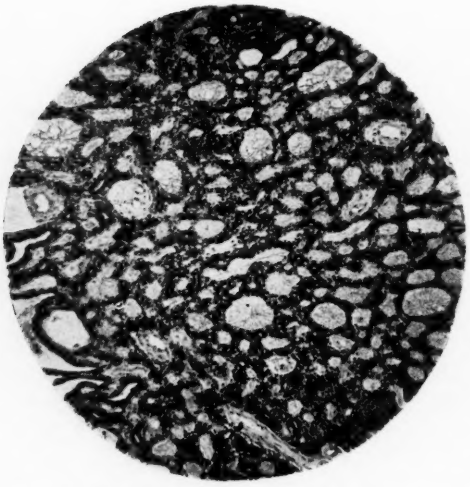

Fig. 2. Photomicrograph of the lesion under low power to illustrate the cell type and general histologic structure. The tumor cells are irregular in shape and undifferentiated. They form solid clumps and cords. In the lower left hand corner the small acini and cysts lined by columnar epithelium may be observed indicating glandular type of growth.

NOTE: Photomicrographs were made by Dr. David Morgan.

The growth appeared to be cured, at least temporarily, after four months of ionization, but in order to make sure, treatments were continued for about a year at intervals of several weeks. No further change could be noted. Treatment was discontinued and I lost track of the patient for five years. I finally located her and examined the ear in my office October 5, 1935. There was no change in the external canal, the scar having the same appearance as seven years ago.

Pathologic report by Dr. Baxter L. Crawford.

Gross description: Specimen submitted for examination consists of a small, irregularly shaped, soft, gray piece of tissue which measures 1.75 cm. x 0.5 cm.

Histology: Examination of sections from the small piece of tissue reveals that it is largely composed of masses of cells, with large deeply stained nuclei, which form solid cords and clumps throughout a small amount of cellular connective tissue stroma. One surface of the tissue is practically covered by stratified squamous epithelium, which is inflamed and ulcerated. The cells comprising the growth vary considerably in different locations, and in areas the cells are closely packed in solid clumps and cords, with a very small amount of cel-

Fig. 3. Photomicrograph of the lesion under high power to illustrate the columnar type of cells lining the glands and small cysts.

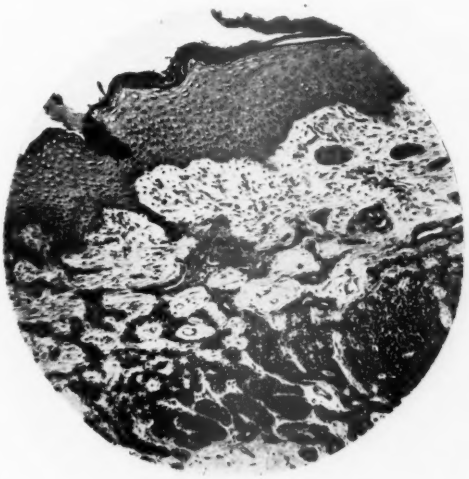

Fig. 4. Photomicrograph under low power to illustrate the squamous epithelium of the surface (normal skin) and the different type of infiltrating epithelium in the deeper structures.

lular stroma; in other places they are distinctly columnar or cuboidal in type and form acini. In these solid areas there are spaces between the cells which seem to contain a mucoid material and many of the acini contain a similar secretion.

The lesion is evidently neoplastic and because of the diffuse infiltration by the epithelial cells it must be considered malignant. From the presence of the columnar type of epithelium and the attempt at acinous formation in areas and the presence of a mucoid secretion, it is considered to be of gland origin, but the exact source of the growth could not be determined from the histology. Because of the adenomatous-like arrangement, the lesion would be considered of relatively low grade malignancy.

Diagnosis: Adenocarcinoma of the external auditory canal.

Treatment: In the cases reviewed, radical surgery was performed and in most instances was followed by irradiation. This treatment was successful, excepting the two cases of long standing. Thorell[1] at the Radiumhemmet, Stockholm, feels that "combined surgical and radiation technic, using electrocoagulation followed by radium, seems to be the method of choice."

The treatment employed in my case is a departure from the accepted surgical procedure and it is not to be understood that I advocate this treatment in preference to radical surgery in cases of cancer of the ear. It was undertaken because the patient was unwilling to undergo radical operation, and while it was justified in this case I do not endorse it as the treatment of choice. However, it suggests a form of treatment that may be used in cases of this kind, provided the neoplasm is primary in an accessible part of the ear.

The treatment used in this case is termed "radio-ionic medication of uranium," by the late Henry F. Harris, who experimented with uranium ionization for several years and who describes its use in treatment of cancer in an unpublished paper, a copy of which I possess.

The treatment is based on the theory that the radio-active ions of uranium are carried into the cells of the tissues by the direct current, where they remain and continue to act for several days after each ionization.

The apparatus used in this case was made for me by Geo. P. Pilling and Sons Co., and was a simple motor-generator with a delivery capacity of thirty-five milliamperes of direct current. It was equipped with a reliable milliampermeter and three potentiometers to control the current. The electrodes were the usual electrodes used in ionization of other chemicals.

The solutions used were a 2 per cent solution of uranium sulphate, a 2 per cent solution of chromium sulphate and a 2 per cent salt solution.

The procedure employed was the same as ionization treatment for other conditions.

The major portion of the growth was removed by snare without local anesthesia. Bleeding was slight and easily controlled by a light packing of the external auditory canal with cotton. Subsequently ionization of uranium was carried out at intervals of two to five days, using an average of four to five milliamperes of current for five to ten minutes at each sitting.

The treatment was painful only on one occasion when the motor became irregular.

CONCLUSIONS.

1. Adenocarcinoma is a rare neoplasm of the ear as reported, but probably occurs much more frequently, which might be shown if biopsies were made on all growths removed from the external auditory canal and reported.

2. Early recognition of the lesion is an important factor in its treatment, as results obtained in three of the five cases shows.

3. Two of the cases were of low grade malignancy as reported by the pathologist.

4. Adenocarcinoma of the middle ear may originate in the external auditory canal, the source possibly being in the sebaceous or ceruminal glands.

5. Ionization of uranium in malignant neoplasms is worthy of further study and may be a valuable addition to the treatment of accessible cancers.

1923 SPRUCE ST.

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XXXVI.

MIXED TUMOR OF THE NASAL SEPTUM: REPORT OF A CASE.*

I. F. Weidlein, M. D.,

Cleveland.

The usual site of mixed tumors is in the parotid region. These tumors rarely appear in the upper air passages and accessory sinuses. They seem to be most uncommon in the nasal septum. In 1932 Stevenson[1] reported a case of mixed tumor of the nasal septum. He stated that careful search of the literature had failed to reveal a report of a mixed tumor of the nasal septum before his report. We have not found any case reported since Stevenson's report.

REPORT OF CASE.

Case 1.—This patient was a 53-year-old white woman, who was first referred to the Otolaryngological Dispensary of the University Hospitals by Dr. William B. Chamberlin on October 9, 1934. She complained of having had severe frontal and temporal headaches since childhood; she claimed that she had never been free of headache. The headaches were dull and pounding in nature, and seemed worse in the morning and evening, and they were usually associated with abdominal distention and a feeling of heaviness in the abdomen. She had vomited at irregular intervals, two or three times a month, for the preceding fifteen years. This vomiting was always associated with headache and pain in the abdomen. She had not vomited during the five months preceding admission to the Dispensary but she had been nauseated. About a year before admission, the left side of the nose had become swollen. This swelling did not subside but grew gradually worse. Six months before coming to the Dispensary she began to have pain in the left side of the face and in the left temporal region. At that time the left side of the nose became blocked and she was unable to breathe through it from then on. During these six months she had had frequent left sided epistaxis. A week before admission she began to have very severe pain in the left side of the nose, face and head accompanied by vertigo and some nausea. She also had

*From the Department of Surgery of the University Hospitals and the Western Reserve University School of Medicine.

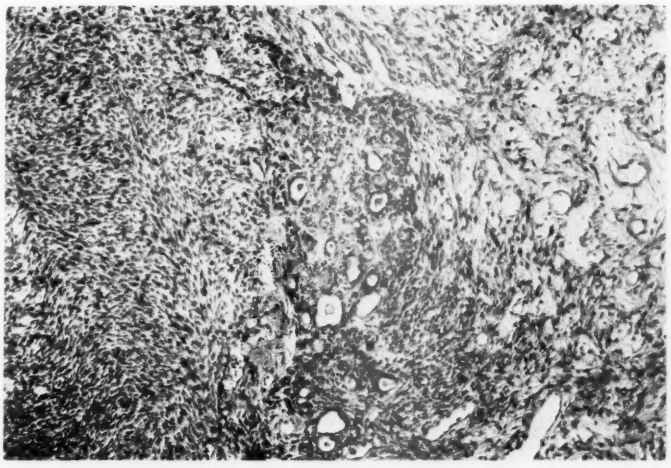

Fig. 1. Photomicrograph showing the marked variation in the histologic structure of the tumor. To the left the tumor is comprised of plump spindle cells. In the center, cells are epithelial in character, are distributed in a hyaline matrix and exhibit alveolar differentiation. To the right the tumor is myxomatous and richly vascularized. x 130.

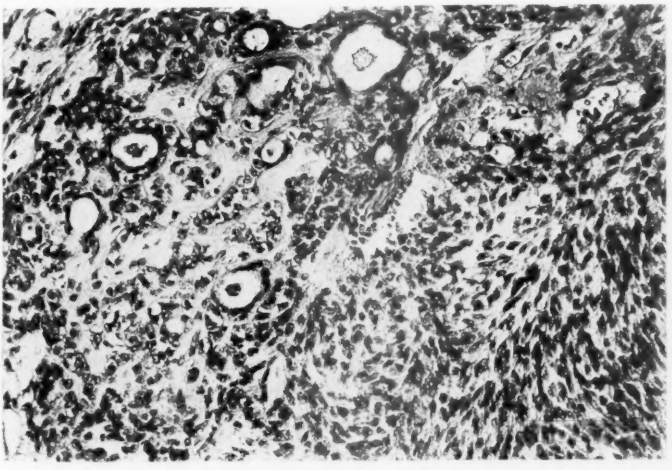

Fig. 2. Higher magnification of portion of tumor showing alveolar differentiation of epithelial cells. x 300.

severe pain in the left eye. There had been very slight tenderness over the left side of her nose for one month.

Her family history and marital history were irrelevant. She had had "ear trouble" since she was young and had had several attacks of influenza. She had experienced shortness of breath on climbing stairs for one year.

She was a well developed, obese woman, mildly but chronically ill. The pupils were equal and regular, and reacted to light and accommodation. The teeth were in poor condition; she had many gold crowns and fillings.

Externally there was a moderately hard bony swelling of the left side of the nose, which was slightly tender. The septum was fairly straight. The entire left nostril was filled with a firm rounded mass. The anterior inferior portion of the mass had ulcerated and was covered with a blood clot. There was a small amount of white discharge in the left side of the nasopharynx. The posterior portions of the septum were visible; also the eustachian orifices and posterior ends of the inferior turbinates were visible in the postnasal mirror. No mass was seen in the nasopharynx.

The tonsils were small and imbedded; the pharynx was slightly injected. The larynx was negative. There was no adenopathy of the neck. Both membrana tympani were dull and retracted. On transillumination the frontal sinuses were clear; both antra were dark.

The roentgenologic report by Dr. Eugene Freedman was as follows: "October 9, 1934. Films of the sinuses show a mottled cloudiness throughout both maxillary sinuses and throughout the right ethmoid cells. The frontal sinuses are also slightly hazy. The findings suggest thickened mucosa."

On October 12, 1934, the nasal growth was removed.

The pathologic report by Dr. Alan R. Moritz was as follows:

"The specimen consists of an ovoid nodule measuring 2.5 by 1.5 cm. A portion of one surface of the nodule is smooth and apparently covered by an intact mucous membrane. The nodule is firm and on section comprised of gray, fibrous appearing tissue which appears to be distributed in the form of interlacing streams and whorls. Almost the entire nodule seems to be comprised of a tumor which, although circumscribed, is not definitely encapsulated.

"Microscopic sections indicate the specimen to be almost entirely comprised of a tumor. The tumor is made up of a variety of cells,

most of which are spindle shaped and arranged in the form of solid masses, the long axes of the cells being parallel to one another. Partially separating these solid masses of cells are anuclear trabeculæ of hyalinized connective tissue. Scattered focally throughout the tumor are collections of small cuboidal or polyhedral cells arranged in solid masses or in the form of tubular alveoli and disposed in isolated groups within an abundant anuclear homogeneous, acidophilic matrix. In some situations the cells appear to occupy lacunæ in this matrix. Mitoses are uncommon and despite the cellular pleomorphism the nuclei are round, chromatic and quite uniform in size. No definite capsule is identified.

"Diagnosis: Mixed tumor (salivary gland type)."

10515 Carnegie Ave.

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XXXVII.

LYMPHOSARCOMA OF THE LARYNX: REPORT OF A CASE.

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PORTLAND, MAINE.

True sarcoma of the larynx is rare. Chevalier Jackson,[1] in a series of 643 neoplasms of the larynx, did not observe one. New[2] reported that since 1910 only six patients with sarcoma of the larynx have been seen at the Mayo Clinic, as compared with more than 800 patients with carcinoma of the larynx. Chevalier Jackson notes that lymphosarcoma of the larynx is rare, and if limited to this structure should be dealt with as other malignant growths, but if the laryngeal lesions are associated with a general lymphosarcomatosis they may be regarded as a minor element in a fatal disease.

This is the report of a case of lymphosarcoma of the larynx associated with skin manifestations, with later metastases to the small bowel with perforation, to one breast and then the other, to the tissues about the left mandible, to the left labia minor, to the tissues about the lower ribs in the axillary line, and to the heart muscle, causing coronary occlusion and death. It is of interest, as the initial lesion was in the larynx.

F. Massei[3] (1899) reports five cases of sarcoma of the larynx with skin manifestations, and remarks that the skin should always be examined in cases of laryngeal tumor and the larynx should be systematically examined in sarcomatosis.

Sarcomata are mesoblastic in origin. There is controversy concerning the classification of these tumors. The leucoblastomata are a subdivision that is sometimes considered to contain Hodgkin's disease on one hand and the lucemias on the other, with the various types of lymphosarcoma taking midway position. None of these groups are entirely fixed, for blood changes may occur in tumors closely resembling Hodgkin's disease and tumors sometimes occur in the course of certain leucemias. Skin manifestations may occur in any of these diseases. Intractable itching is a frequent complaint and is probably due to a deposit of abnormal lymphoid cells about the sensory nerve endings in the skin. Macular and papular rashes are not infrequent and multiple skin tumors sometimes occur, certain of these latter are termed mycosis fungoides.

The lymphoblastomata are systemic conditions, and while one may speculate as to whether they arise from a single focus in a given case or whether some urge stimulates abnormal change in all mesoblastic tissues, the result is the same—multiple metastasis and death.

REPORT OF A CASE.

CASE 1.—H. J., female, aged 44, was first seen on March 25, 1934. She complained of difficult, painful swallowing as food passed from the hypopharynx into the upper esophagus. This had been increasingly noticeable for the prior three months, during which time she had lost twenty pounds in weight. She dated her difficulty from a food poisoning which occurred after eating shredded codfish some three months before.

Her general health had always been good, and her past history was negative except that she had had a supravaginal hysterectomy for an intramural fibroid three years before.

Her family history was unessential except that her mother died from tuberculosis at about the time of the patient's birth.

Examination of her ears, nose and throat revealed no abnormality except that a mass about one inch wide and a quarter of an inch thick was visible behind her arytenoids and there was a slight edema of her left arytenoid. The color and appearance of this growth resembled a portion of the lip of a normal cervix uteri. The larynx was otherwise normal in appearance and function. A copper colored macular rash was noted on the flexor surfaces of her arms and legs.

Four days later she was sent to the Maine General Hospital for further study. Her general physical examination, except for the rash noted above, a blood Wassermann and Kahn, an x-ray examination of her chest and a tuberculin skin test were negative. A fluoroscopic study of her swallowing function suggested a postlaryngeal obstruction. The dermatological service felt that the skin lesions resembled a luetic rash. It was thought that the postlaryngeal swelling might be due to granulation tissue about a fish bone that had lodged at the time of the fish poisoning from which she dated her difficulty.

On March 24, 1934, a direct laryngoscopy and esophagoscopy were performed. When the direct laryngoscope was introduced the upper portion of the mass resembled the mirror view, but when the larynx was lifted forward an eroded area was observed on its posterior surface, no fish bone was seen or palpated and a specimen was taken for examination, which was diagnosed as granulation tissue by the pathologist.

The pathologist's report strengthened our suspicion that the postlaryngeal mass was granulation tissue caused by an impacted fish bone, and on April 4, 1934, a second direct laryngoscopy was performed. No bone was found and another specimen was taken which again was reported as granulation tissue.

On May 5, 1934, she was referred to another institution. At this time the tumor mass appeared as before, but the arytenoids were more swollen and almost obliterated the pyriform sinuses. She was becoming slightly hoarse and the copper colored macular rash was more pronounced. While she was at this institution her physical examination, with the exceptions noted above and the fact that the margins of her liver were palpable two fingers' breadth below the costal margin, x-ray of her chest, blood and platelet count, blood and spinal fluid, Wassermann and Hinton, and spinal fluid examinations were reported to be negative. She was seen in consultation with the dermatologists, who felt that her rash might possibly be due to lues, and suggested that she be treated with bismuth and neosalvarsan for a period of time and her blood be checked each week. On May 28th she was discharged unimproved with the suggestion that the antiluetic treatment be continued.

In July, 1934, she was readmitted to the Maine General Hospital because of urgent dyspnea, and a tracheotomy was performed. Her anti-luetic treatments had been continued for two months, and her blood Wassermann and Kahn were still negative. X-ray studies of her chest, blood examinations, repeated sputum examinations and tuberculin tests threw no additional light on her condition. The dermatologic service offered the opinion that her rash might be due to an erythema multiforme. On August 8, 1934, another direct laryngoscopy was performed. At this time her arytenoids were so swollen that they practically occluded her larynx, although a fleeting glimpse of the cords showed them to be unaffected. (Her voice was thick but not particularly hoarse.) Another specimen of tissue was removed. The pathologist noted a cellular tumor of lymphoid cells fairly regular in size on a scanty fibrous tissue stroma and felt that this should be classified as a new growth and not as granulation tissue, making a diagnosis of lymphosarcoma.

Deep x-ray treatment of her laryngeal region was begun on August 17, 1934, and she received five treatments of 300 R units each. As a result of this treatment, the arytenoid swelling and the postlaryngeal mass rapidly disappeared. The tracheotomy tube was removed on August 24, 1934, and she was discharged from the hospital on August 29, 1934.

On October 28, 1934, a specimen of tissue from a skin lesion was reported by the pathologist to be lymphosarcoma and the dermatological service then felt that her rash was akin to mycosis fungoides.

In November, 1934, at weekly intervals, she was given deep x-ray treatments of three-fourths of an erythema dose to her entire skin surface, with the result that the macular rash became less noticeable.

On November 14, 1934, a nodule was discovered in her right breast. This was given two treatments of 300 R units each.

In course of the next four months her larynx became normal in appearance and function. Her general health was good and she did her work and went about as usual. Her rash again became more pronounced, and in January, 1935, she was again given general deep x-ray to her skin.

On January 2, 1935, a nodule was noted in the left breast and this was also given two treatments of 300 R units each.

On January 31, 1935, she awoke with severe general abdominal pain without nausea and was readmitted to the Maine General Hospital—for the previous few weeks she had noted indefinite bladder symptoms—her abdomen was ridged. X-ray examination revealed no gas below the diaphragm nor in the small bowel. It was felt that she had an intestinal obstruction. She was operated upon that afternoon. The lower abdomen and pelvis was filled with a thin pea-soup-like pus; further exploration discovered a loop of small intestine adherent to the bladder. In a ring-like growth, about two inches in length in the posterior surface of the intestinal loop, was a perforation of sufficient size to admit a finger. The tumor mass was resected and a lateral anastomosis was made. Her recovery was uneventful.

On March 6, 1935, her right breast was amputated under a local anesthesia. The mass in this breast had never totally receded under x-ray treatment and had begun to increase in size. The surgical service felt that the simple removal of the breast was a relatively minor procedure and were curious to discover if the tumor was of the same character.

The pathologist reported that both the tumor mass from the small intestine and from the right breast were lymphosarcoma.

On March 15th she was discharged from the hospital. For the next four and a half months she enjoyed surprisingly good health, although her skin lesions increased in size and became more papular.

On July 25, 1935, a lump was noticed under her left jaw. This was given three deep x-ray treatments of 300 R. units each and subsided.

On August 7, 1935, she was readmitted to the hospital because of a growth the size of a walnut in the left labia minora. This was excised by the gynecologic service and again found to be a lymphosarcoma.

She was discharged from the hospital on August 18, 1935, and subsequently was given three deep x-ray treatments of 300 R units each over her pelvic region.

On November 12, 1935, a mass was noticed on the lower ribs in the left axillary line. Between November 14th and 18th she was given two deep x-ray treatments of 300 R units.

On November 23, 1935, she appeared to be in good health and was enjoying herself at a birthday party when she suddenly stiffened and died.

An autopsy was performed. The anatomic diagnosis was fibrinous pericarditis, metastatic tumor of the heart, occluding the coronary artery by pressure, metastases to the liver, left breast and skin.

Microscopic diagnosis was lymphosarcoma, adult cell type.

The occlusion of the coronary artery was caused by a metastatic tumor in the heart muscle compressing the left coronary artery at its division with the circumflex and interventricular branches. While this occlusion would have occurred in course of time, it is interesting to speculate as to the effect of the deep x-ray therapy given to the mass in the region of the lower left ribs some five days before her death.

The larynx presented no gross or microscopic anomaly.

49 DEERING STREET.

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XXXVIII.

CONGENITAL BONY ATRESIA OF THE RIGHT POSTERIOR NARIS.*

HAROLD LESLIE KEARNEY, M.D.,

NEW ORLEANS.

Congenital atresia of the posterior nares is noteworthy because of its rarity; for its serious nature to the newly born infant when it is bilateral; and because it deprives its victim of the protective benefits of nasal respiration.

The obstruction in congenital choanal atresia may be osseous, membranous or a combination of the two.[2 4 5] The osseous variety is most frequently encountered. It may be unilateral or bilateral. The majority of the reported cases are unilateral and the right side is more commonly involved. The deformity is more frequently encountered in females than in males. Richardson[2] has pointed out that many of the bilateral cases probably die soon after birth under the diganosis of asphyxia of the newly born.

Stewart[4] has reported two cases of bilateral choanal atresia in sisters, aged eighteen and fifteen years, and he suspected from the family history that the same condition had been present in their brother who had died of asphyxia at the age of two days. Heredity is not believed by Stewart, nor by most of the other writers on the subject, to be a factor in its production.

Thomson[5] says choanal atresia is a vestigial structure, being a persistence of the bucconasal membrane. Stewart,[4] after thoroughly investigating the embryology of the region, gives the following summary: "There are three degrees of congenital choanal atresia, which are due, respectively, to: (a) persistence of the buccopharyngeal membrane, excessive growth medially of the horizontal and lateral processes of the palate bone and persistence of the nasobuccal membrane; (b) excessive growth medially of the horizontal and lateral processes of the palatal bone and persistence of the nasobuccal membrane, and (c) persistence of the nasobuccal membrane" J. Parsons Schaeffer[3] believes persistence of the bucconasal membrane or organi-

*Read before the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society at Jackson, Miss., January 18, 1936.

zation of epithelial plugs, which occasionally block the choanæ earlier in fetal life, to be causes of choanal atresia.

The patient with unilateral atresia of the nares may suffer little from nasal obstruction if he has good ventilation of the uninvolved side, his chief discomfort being inability to blow secretions from the affected side. The majority of unilateral cases come to the doctor between the ages of twelve and twenty years, having themselves observed a defect which had been disregarded or overlooked at a more tender age by their parents. Grove[1] has reported a case of congenital atresia of the right posterior naris in a female patient who sought relief at forty-eight years of age. Many of the reported cases had previously sought medical attention, and the prior consultants had done adenoidectomy or turbinectomy, or removed polypi without recognizing the congenital deformity.[4 5]

Richardson[2] observed that "more or less involvement of the middle ear is noted, although a number of cases have been observed in which there was no involvement of the auditory apparatus." In the case here reported the ears were normal. In another of my patients, who gave a history of having had an operation for congenital choanal atresia, done by the late Dr. R. C. Lynch, there was a chronic mastoid infection for which I did the radical mastoid operation.

REPORT OF A CASE.

CASE 10.—Miss C. V., a white female, age thirteen years, was seen in the outpatient department of the Touro Infirmary September 7, 1926. Examination of the nose at this time showed the right side filled with a quantity of thick clear mucus. There was a deviation of the septum to the right. The ears and throat were negative. No obstruction of the nose was visible by anterior rhinoscopy. A probe inserted in the right side of the nose could not be passed into the nasopharynx because of a firm obstruction at the posterior naris. The mother said the child had had obstruction of the right side of the nose since birth. A diagnosis of congenital atresia of the right posterior naris was made and operation advised to correct the condition.

September 8th, under ether anesthesia, the finger introduced into the nasopharynx located the obstruction as a smooth wall joining the septum perpendicularly, about 2 mm. in front of its posterior edge. Examination with the postnasal mirror confirmed this and also revealed the left posterior naris as normal. A probe introduced into the right nostril met with firm resistance at about the location of the posterior naris. No opening was found in this partition. A small

curette was then introduced along the floor of the nose and was forced through the partition until it encountered the finger introduced into the nasopharynx. The partition at this point was bony and thin. The curette was then forced through the partition at the upper part of the posterior naris until it encountered the finger in the nasopharynx. The partition at this point was also bony and thin. Between these two openings the partition was composed of thicker bone. A scissors was then introduced with one blade in each opening and, guarded by the finger in the nasopharynx, the heavy structure was cut. A rasp was then used to enlarge the opening to the normal size of the posterior naris. An adenoid forceps was then employed to bite off a portion of the posterior edge of the vomer, as recommended by St. Clair Thomson. A one-inch strip of iodoform gauze was then introduced in the right side of the nose, carried across the posterior edge of the septum and brought through the left side of the nose where the two ends were tied together. Two days later a fresh strip of iodoform gauze was attached to the previous strip and was pulled into place as before. This procedure was very painful to the patient and was abandoned at subsequent dressings, at which a cotton wound applicator was passed through the opening. The method was unsuccessful, and the opening had closed about three weeks after the operation.

In reviewing the probable causes of failure, it was thought that the deviation of the septum may have been a factor, and submucous resection was done October 6th. The right posterior naris was reopened and the vomer was removed all the way back to the posterior edge of the septum. Both sides of the nose were packed with vaselined gauze.

The postoperative course after the second operation was entirely satisfactory, and three months later the patient was discharged with perfect nasal respiration and greatly pleased at being able to blow the right side of her nose as well as the left.

SUMMARY.

A case is presented of osseous, unilateral, congenital choanal atresia in a female, aged thirteen years, in which the deformity occurred on the right side. This is a typical case of congenital choanal atresia in that the condition is most frequently osseous, more commonly is unilateral, most frequently affects the right side and occurs more often in females.

1403 DELACHAISE STREET.

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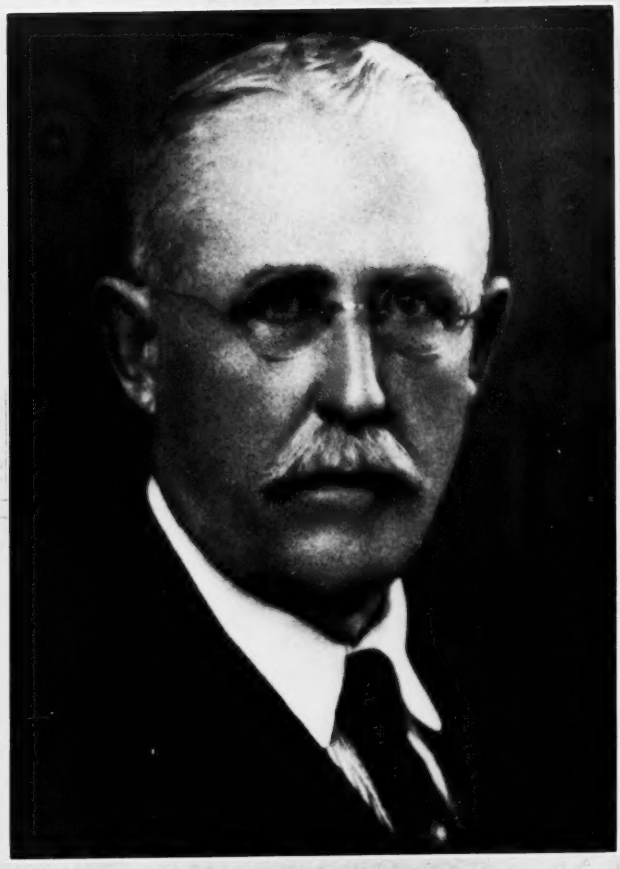
EDWARD BRADFORD DENCH

In the death of Edward Bradford Dench, on February 21, 1936, American Otology lost one of its conspicuous leaders. Indeed, it may be said that in view of his many important contributions to the specialty, oral as well as written, for over forty years, he may be regarded as its *most* conspicuous representative.

Dr. Dench was born at Leedsville, New York, January 16, 1864. He was the son of Joseph Bradford Dench and Frances Lester Dench of Fitchburg, Mass., and a descendant of Governor Bradford of the Plymouth Colony and of Captain Robert Dench who came to this country from England in 1715. Dr. Dench, after attending the Bridgeport, Conn., High School, graduated from Yale Scientific, with the degree of Ph.B. in 1883, and from the College of Physicians and Surgeons, Columbia University, with the degree of Doctor of Medicine in 1885. Following his graduation he entered St. Lukes Hospital as intern, where he remained for two years, from there going to the Hudson Street branch of the New York Hospital, where he remained for a like period of time. In 1889 he became connected with the New York Eye and Ear Infirmary in the capacity of Assistant Surgeon. He soon became Surgeon and continued as such until his retirement in 1929. He founded the Otological Clinic of St. Lukes Hospital and in 1914 established the department of Otology at St. Lukes, where he was consulting and attending otologist until 1929. At the time of his death, he was consulting otologist to the New York Eye and Ear Infirmary and to the New York Orthopedic Dispensary and Hospital. Dr. Dench was for many years professor of Otology in the University and Bellevue Hospital Medical College.

He was an enthusiastic and faithful member of the various local, national and international societies connected with his specialty and always could be depended upon to contribute to their programs in a manner that was both instructive and authoritative. He was one of the founders and was the first President of the American Laryngological, Rhinological and Otological Society in 1896, and in 1931, in recognition of his distinguished achievements, was made Honorary President of the Society. He was a founder and former President of the New York Otological Society, 1904-1905. He became a member of the American Otological Society in 1892 and was its President in 1910. He was honored by election to membership in the Royal Society of Medicine, London, and in the Collegium Oto-Rhino-Laryngologicum Amicitae Sacrum. His face was a familiar one at the meetings of the Section of Otology of the New York Academy



Edward B. Deuch

of Medicine, of which he was a former Chairman. During the World War he held the rank of Major in the Medical Reserve Corps in the Army and was active at Camp Upton during the enlistment period.

He married Marie Antoinette Hunt of Boston, October 3, 1888, and had a daughter, Mrs. J. Russell Hawks of Detroit, both of whom survive.

Dr. Dench was the author of a widely known and used book on the Diseases of the Ear, which first appeared in 1895 and which went through five editions, the last in 1919. The number of contributions made on the subject of Otology by Dr. Dench exceeds that of any other one in this country. Not merely were they numerous but many of them were of outstanding values. He read twenty papers alone before the American Otological Society, of which the first, read in 1894, was entitled "Middle Ear Operations as a Means of Improving the Utility of the Organ of Hearing," and was an exhaustive study of operations inside the drum with the indications for their employment and the technic employed. Two years later, he reported a case of thrombosis of the lateral sinus, following suppurative otitis media, cured by operation. We have not taken occasion to look up the statistics, but this must have been one of the earliest cases reported in otologic history, certainly in this country.

In 1899 he reported seventeen cases of chronic otorrhea in which he had performed the Stacke operation, thirteen of these entirely cured and four improved. This was undoubtedly one of the earliest papers in American literature on the radical operation. In 1901 he reported a case of otitic brain abscess cured by operation. This was one of the first successful operations for brain abscess reported by an otologist in America. Dr. Dench possessed not merely the diagnostic skill, but also the surgical ability to operate in such cases. In a comprehensive paper, read in 1902, he called attention to "The Value of Bacteriological Examination of Discharge in Acute Otitis Media as Determining the Necessity of Operative Interference." Unlike many others, he did not hesitate to report his non-successes. In 1905 he reported two cases of brain abscess, carefully operated upon, but which resulted unsuccessfully, and in 1906 a case of cerebral abscess following chronic suppurative otitis media, operation and death. An exhaustive paper on Brain Abscess appeared in 1907. In 1908 Dr. Dench reported a case of acute suppuration of the labyrinth, following acute otitis media, operation, recovery. He studied carefully the Haynes method of draining the cisterna magna for otitic meningitis and in 1913 reported three cases treated by the method without suc-

cess. A year later he contributed a paper to the proceedings of the Society on "The Treatment of Accidental Wounds of the Dura During Operations Upon the Mastoid Process With a Report of Several Cases." Dr. Dench was a pioneer in the use of a graft in the radical mastoid operation, and in 1915 contributed a paper on that subject. He was an enthusiastic supporter of the investigation of Progressive Deafness instituted by the American Otological Society, and at the time of his death was a member of the special committee having the work in charge. In 1921, in a paper on "Operative Procedures Upon the Internal Jugular Vein in Cases of Sinus Thrombosis of Otitic Origin," he discussed in a masterly way the operation, and in 1925 he presented an authoritative paper before the Society on Labyrinthine Surgery.

To the American Laryngological, Rhinological and Otological Society, of which he was the first President, he contributed no less than twenty-five papers, extending down almost to the year that he was stricken. Of these mention should be made of a paper presented in 1899 on the Technique of Synchiotomy for improving the hearing in otitis media chronica purulenta, with improvement in twenty-five out of twenty-six cases. He was one of the first to advocate ligation of the internal jugular in septic thrombosis of the lateral sinus, and in 1900 reported three cases where he had successfully tied that vein. This was followed two years later by an excellent paper on the same subject. At the meeting of the Society, in 1903, he contributed a paper giving a complete description of the radical mastoid operation and recommending the use of a Theirsch graft. In 1907 he reported three cases of suppurative labyrinthitis cured by operation upon the labyrinth, a further example of his pioneer work. In 1912 he was among the first otologists to report two successful operations on brain abscess. In a paper read three years later he again discussed the value of the labyrinthine operation. We have not, on account of time, included any papers that he read before the International Congresses. His contributions were always timely and in many instances represented views and work well in advance of most of his confreres, as, for instance, his ligation of the jugular vein in lateral sinus thrombosis.

Dr. Dench was a close observer and an indefatigable worker. Some phase of otology was in his mind during all his waking hours. Indeed, to the question he was once asked, "What is your hobby?" he replied "otology."

Dr. Dench's preparation for his life work can well be followed by the young man of today. As far back as when he was a student

at Yale, he did original work in otology, particularly in the microscopic anatomy of the ear. That was fifty years ago, when few, if any, otologist had any acquaintance with the subject.

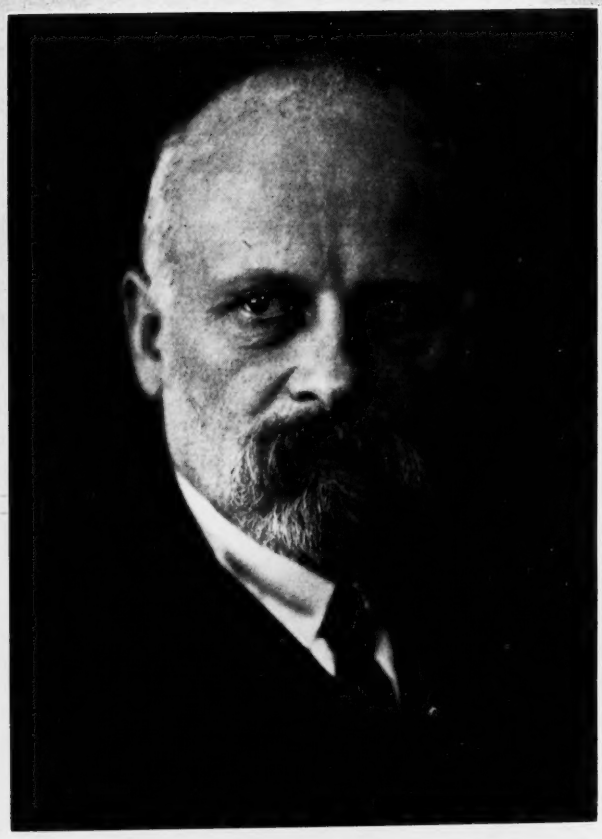
Today the importance of an accurate knowledge of the anatomy, physiology and pathology of the ear is being more and more generally recognized. Indeed, the granting of the certificate of the American Board of Otolaryngology is dependent to a considerable degree upon possessing such an acquaintance.

While still active in his specialty, Dr. Dench was stricken in 1929 and was from that time until the time of his death a constant sufferer. Through it all, his mind remained clear and he continued to keep in touch with all that concerned the science of otology. The courage and patience displayed by him during his long illness call forth only words of praise and admiration. To him fittingly can be applied the lines of Robert Browning—

One who never turned his back but marched breast forward;
Never doubted clouds would break;
Never dreamed, though night were worsted wrong would triumph;
Held we fall to rise—are baffled to fight better, sleep to wake.

THOMAS J. HARRIS.

A memorial before the New York Academy of Medicine.



L. Barany

ROBERT BÁRÁNY.

1876—1936.

Robert Bárány died on April 8th, 1936, after having been ill for years following an apoplectic stroke.

He was born April 26, 1876, in Vienna. He matriculated in 1894 at the Medical School in that city where, in 1900, he obtained the degree of M.D. He then studied internal medicine for one-half year with von Noorden at Frankfurt and psychology with Kraepelin in Heidelberg and with Pfi at Freiburg. After one year's surgical training at the General Hospital in Vienna, he was appointed to the Ear Clinic in Vienna with A. Politzer, in 1905, and there acted as demonstrator and amanuensis until, in 1909, he was made instructor in diseases of the ear at the University of Vienna. In 1917 he became instructor in otorhinolaryngology at the University of Upsala and the same year obtained a professor's name, honor and dignity and was that same year made teacher in eye, ear, nose and throat diseases and chief for the university otological clinic.

In the meantime he had become a Swedish citizen.

He was correspondent and honorary member of a great many academies and learned societies and obtained many scientific distinctions, among them the Politzer prize at the International Otological Congress in Boston in 1912, and the Erb medal in 1913, the Guyot prize from the University of Groningen in 1914 and the Nobel prize in physiology and medicine from the Karolinska Institute in 1915. He was Honor Doctor at the Karolinska Institute in 1924, and was made Commander of the Northern Star of the First Class in 1927.

He leaves a widow and three children, the sons—Ernst and Franz and the daughter, Ingrid—all three studying medicine at the University of Upsala.

While still quite a young doctor, Bárány became known for his work on the vestibular apparatus. His graduate thesis, "Investigations on the Rhythmical Nystagmus Which Is Created by Reflexes From the Vestibular Apparatus of the Ear and Its Associated Phenomena," founded and confirmed his world reputation. In this remarkable work, which to the greatest extent is based upon careful clinical observation, Bárány communicates an amazing multitude of observations and amongst those the ones which laid the foundation

for the caloric reactions, and he brought the multitude of partly known and often apparently contradictory facts under a uniform point of view, thereby throwing new light on the entire labyrinthology and adds to that the caloric reaction and its essential clinical applications. This work was complemented by some following works on clinical investigations of the larynx and caused intensive work amongst otologists all over the world, and it became the basis for a great deal of literature. Often and from a variety of sources I have heard the view expressed that all the scientific knowledge which we have today regarding the vestibular apparatus is contained in this graduation thesis which is now thirty years old.

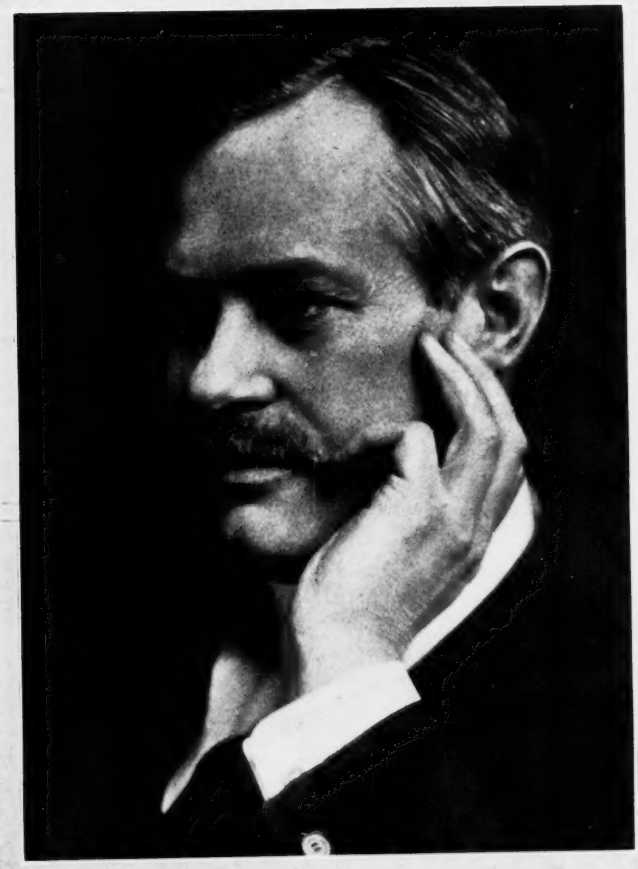
As physician and surgeon at the war hospital in the fortress of Przemysl Bárány came, during the World War, in contact with a great many skull injuries. After the fortress had fallen and Bárány, through the intervention of Prince Carl of Sweden, had been liberated from Russian captivity, he worked with great energy that his experience with the closed treatment of skull injuries should benefit the victims of the war.

In his private life, Bárány was retired, quiet and friendly. His interests were his home, family and music. Transplanted, as he was, to surroundings which for him were strange, far from his home, family and all old friends, and with the comparative isolation which follows transplantation from the center to a periphery, he retired to himself and became strange to many amongst his new surroundings. A heavy shadow which long rested on him was a desire for his beloved Vienna, and it was not without bitterness that he contemplated that his own country had not been able to find a place for him. As the years went on, his clinics, his patients, and above all, his research, became the interests which overshadowed everything else. Idealism, a burning interest and a tireless energy which constantly distinguished him, make him also in this respect a great example. Through overwork, perhaps, he caused the illness (hypertension) which, after repeated strokes, ended his brilliant and still lonesome career.

For his sixtieth birthday, on the 22nd of April, an international celebration on a considerable scale had been planned. At the invitation of all the editors of Acta Oto-Laryngologica, several hundred otologists from all parts of the world sent Bárány greetings, and they had contributed to a collection which, under Bárány's direction, was to have been used to further otology. But Bárány did not live to see this day of honor. He died in Upsala on the 8th of April and was cremated and buried at the Stockholm crematory.

Amongst the many wreaths at his bier was one upon the white ribbon of which read "To Robert Bárány, the pioneer research man. Gratefully from otologists from the entire world." The writer placed it there with the following words: "For your sixtieth birthday, which was soon to come, otologists from all parts of the world wanted to do you honor. Several hundred greetings had already reached us, addressed to the pioneer in clinical research on the labyrinth, to the greatest otologist of our generation. More than anyone now living have you contributed to the development of our science. Your restless, burning spirit saw clearly where the rest of us were groping in darkness. Your power of creation gave us new means and broke new roads for work in our action to relieve sufferers without number. In appreciation of your research, in gratitude for what you gave, uncounted numbers of otologists from all over the world lay these flowers at your tomb. Peace to your memory."

GUNNAR HOLMGREN.



Arthur B. Duel.

ARTHUR B. DUEL

Doctor Arthur B. Duel, vice-president and chairman of the Board of Surgeons of the Manhattan Eye, Ear and Throat Hospital of New York, died April 11, 1936, at his country estate, "Laurelwood."

Doctor Duel will be remembered by future generations chiefly for his experiments, conducted jointly with Sir Charles Ballance of London, whose death preceded his own by only two months. The final report of this work, which was undertaken by the two scientists at Doctor Duel's country home in 1930 and resulted in a practical method of anastomosing the severed ends of the facial nerve to relieve facial paralysis, appeared as the leading article in the March (1936) issue of the Annals. Associated with them in the work was the co-author of this article, Doctor Thomas G. Tickle.

Doctor Duel was born in Granville, N. Y., December 14, 1870. the son of Hiram D. and Almera J. Hicks Duel. From the University of Vermont Doctor Duel went to the Harvard Medical School, graduating cum laude in 1894.

From the commencement of his practice in 1895 until his death he occupied various positions with the Manhattan Eye, Ear and Throat Hospital. From 1896 until 1900 he was connected with the New York Eye and Ear Infirmary, and from 1908 to 1913 he was Professor of Otology at the New York Polyclinic Medical School and Hospital.

Doctor Duel was a former vice-president and a trustee of the New York Academy of Medicine and was chairman of the building committee of the Academy when its present building at Fifth Avenue and 103rd Street was erected. He was at the time of his death and had been for many years chairman of the board of trustees of the Research Fund of the American Otological Society.

Dr. Duel was a former president of the American Otological Society (1927) and a member of the American Laryngological, Rhinological and Otological Society, Medical Association of the City of New York, the New York Otological Society and the American Medical Association and the American College of Surgeons. His foreign associations included membership in the permanent committee of the Congrés Internationale d'Oto-Rhino-Laryngologie, the International Collegium Otolaryngologicum, and an honorary fellowship in the Société de Laryngologie des Hospitaux de Paris. He was a Knight Commander of the Crown of Rumania.

He is survived by his widow, who was Mary Kay Crawford, and two children, Mary Hobart Duel and Arthur B. Duel.

Books Received.

Allergy of the Nose and Paranasal Sinuses. A Monograph on the Subject of Allergy as Related to Otolaryngology.

French K. Hansel, M.D., M.S., Assistant Professor of Clinical Oto-Laryngology, Washington University School of Medicine. Cloth. Octavo of 820 pages with 58 illustrations and three color plates. St. Louis: The C. V. Mosby Company; 1936. Price $10.00.

At this time, when so much that is authentic—and so much more that is conjectural—has been written on the subject of allergy, the rhinologist has great need of a sober and informative manual to guide him. Hansel's monograph amply fills his requirements. The author's mature experience in the field of allergy combined with the painstaking care that has been given to its preparation should make this the dominating text on the subject for some time to come.

The first chapters are devoted to the Physiology, Biochemistry and Bacteriology of the nasal tissues and secretions. Next are developed the fundamental phenomena of allergy and their manifestations in the nose. Following are descriptions and evaluations of the various tests and the many theories and practices of treatment. Hay fever is discussed by itself in detail.

While the author's own views characterize the book and give direction to whatever conclusions are expressed, this work is essentially a compendium of the entire published work on Nasal Allergy. Authorities are freely quoted. Planned as a reference work, the material is well arranged and accessible; an altogether satisfactory book.

Diseases of the Nose, Throat and Ear for Practitioners and Students.

Edited by A. Logan Turner, M.D., LL.D., F.R.C.S.E., Consulting Surgeon, Ear and Throat Department, Royal Infirmary, Edinburgh, with the collaboration of J. S. Fraser, M.B., F.R.C.S.E.; Douglas Guthrie, M.D., F.R.C.S.E.; Charles E. Scott, M.B., F.R.C.S.E.; J. D. Lithgow, M.B., F.R.C.S.E., G. Ewart Martin, M.B., F.R.C.S.E., and John P. Stewart, M.D., F.R.C.S.E. Cloth. Octavo of 473 pages with 243 illustrations and 8 color plates. *Fourth Edition*, Revised and Enlarged. Baltimore: William Wood and Company: 1936. Price $6.00.

This edition, following close upon the heels of the last, is proof of the well-deserved popularity of this work. It is a most satisfactory text. Not a word nor an illustration is wasted. The information is comprehensive and concise; ample consideration is given to anatomy and pathology; the diagrams and illustrations are of a high order.

Each section has been revised and a short chapter on Allergy has been added.

Notices.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Kansas City, May 9, 1936, during the meeting of the American Medical Association. Forty-three candidates were examined, of which number thirty-four were passed and nine were conditioned or failed.

The Board desires to announce that hereafter examination will be of two days' duration, including written as well as oral tests. Furthermore, after January 1, 1938, no certificate will be granted "formally"—that is, without examination, and all candidates shall take the pathology examination.

An examination will be held in New York City, September 25 and 26, just prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. Prospective applicants for certificate should secure application blanks from the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Neb.

W. P. WHERRY, M.D., Secretary-Treasurer.

H. P. MOSHER, M.D., President.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The American Academy of Ophthalmology and Otolaryngology will meet in New York City, September 26th to October 3, 1936. The headquarters will be at the Waldorf Astoria Hotel.

DR. W. P. WHERRY,
Executive Secretary-Treasurer.

Abstracts of Current Articles.

NOSE.

Rhinoscleroma—Is It an Indigenous Disease?

Chamberlin, Wm. B. (Cleveland), Arch. Otolaryng., 23:285 (March), 1936.

1. Scleroma, or rhinoscleroma, is no longer confined to a fairly definite locality in southeastern Europe but is fairly well distributed over the entire world.

2. The bacillus of Frisch is easily obtained in all cases in practically pure culture. This bacillus is very similar to the bacillus of Friedländer and of Abel. Although typical local lesions can be produced by injecting it into animals, it fails to satisfy the postulates of Koch, in that the disease itself cannot be reproduced in animals or in man.

3. The culture of the bacillus of Frisch is not necessary for the absolute diagnosis of the disease. Necessary, however, is the presence of the vacuolated lace or foam cells of Mikulicz and the hyaline bodies of Unna.

4. The mode of transmission or contagion is still unknown, and until this is discovered no definite means of combating the disease will be available.

5. At the present time roentgen radiation or radium in proper dosage offers the best hope of control or cure.

TOBEY.

Research Report on Experimental and Clinical Sinusitis.

Fenton, Ralph A., and Larsell, Olof (Portland, Ore.), Arch. Otolaryng., 23:18 (January), 1936.

Five years of investigation into the histopathology of experimentally produced sinusitis, with clinical application of certain data thus secured, have convinced the authors that owing to the defensive factors inherent in sinus epithelium and the connective tissue elements of its tunica propria almost every preparation applied to the surface of such membranes becomes an irritant unless its strength is isotonic. Solutions stronger than normal are almost certain irritants, as are those which in any way interfere with ciliary action, no matter how well they may function as antiseptics in a test tube. Only a few chemical substances are favorable to the growth of cells and stimulate

an influx of reparative histiocytes and leucocytes. An index of various substances and agents used during the series of studies with the results is appended to this report, and also a note detailing the technic of diathermy for sinusitis, as reported last year.

This year they have demonstrated that histiocytes take up particulate matter from the outer (bony) side of the sinus membrane as well as from the lumen of the cavity, and that such matter is carried widely through the subepithelial tissue within 72 hours of its introduction, pointing to the likelihood of similar dissemination of bacteria in histiocytes.

They do not think that further histopathologic study of sinus membranes of cats treated by the application of solutions to the surface will be profitable at present. Perhaps biochemists, physiologists and bacteriologists, with more extensive equipment and opportunities, may find it possible further to develop some of the lines of investigation which have opened during this and preceding years.

The anatomic problems of lymphatic drainage of the sinuses and of their autonomic innervation remain unsolved. These are difficult and delicate problems, but are nevertheless of much importance in the interpretation of the physiology and pathology of the sinuses.

TOBEY.

LARYNX.

The Superior Laryngeal Nerve and Laryngeal Motility (Nervio laringo superior y motilidad laringea).

Regules, Elias (Montevideo), Ann. O., R., L., Uruguay, 5:3-139, 1935.

Basing his findings upon direct inspection of the larynges of anesthetized dogs subjected to electrical stimulation of the nerves, Regules points out that the prime function of the larynx is the protection of the lungs against entrance of foreign matter. He denies the validity of Semon-Rosenbach's law; varying types of recurrent paralyses are explicable through individual variations in the peripheral distribution of the motor nerves of the larynx.

The superior laryngeal is of capital importance in starting the centripetal reflex of laryngeal closure, which is effected as to the pharynx by the glossopharyngeal and as to the larynx by the recurrent of the same side. Hence destruction of the superior laryngeal as an analgesic measure renders the patient liable to inhalation pneumonia.

FENTON.

PHARYNX.

A Case of Pharyngeal Intubation (Un cas de tubage pharyngé).

Leroux, Louis, Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

The author reports the case of a four-year-old, hyperthyroid child who, in the course of an acute inflammation of hypertrophied tonsils, developed a severe dyspnea with cyanosis and collapse. Direct laryngoscopy revealed a normal larynx and at the same time re-established a good airway. Inspired by the recent publication of the Jacksons on the role of the tongue in asphyxia, a Mayo tube airway was inserted and kept in place the entire night with complete relief. During each of the three succeeding days the child was fairly comfortable, but each night it was necessary to reinsert the airway in order to prevent an impending asphyxia. Recovery occurred when the tonsillitis subsided.

KLAWANS.

ESOPHAGUS, TRACHEA AND BRONCHI.

Foreign Body Transfixing the Esophagus. Peri-Esophageal Abscess. Removal by Esophagotomy. Recovery (Corps étranger transfixant l'oesophage. Abcès peri-oesophagien. Extraction par oesophagotomie externe. Guérison).

Aubin, A., Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

In this case a radiotranslucent foreign body in the esophagus, a pork bone, demonstrated under the fluoroscope by deflecting and splitting the barium meal, was not found on esophagoscopy done thirty-six hours after the accident. Forty-eight hours later the patient returned with dysphagia, marked general signs, and slight swelling in the neck. An esophagotomy was done, a peri-esophageal abscess found amidst peri-esophageal adhesions, and the perforating foreign body extracted. Convalescence was uneventful.

The author emphasizes the value of barium in revealing radiotranslucent foreign bodies. He stresses fever, general symptoms, and swelling of the neck as indications for external operation. He regards the operation as relatively simple.

Although most esophageal perforations lead to mediastinitis, the author feels that the small size of the puncture, and its location not in the posterior wall, favors localization in the peri-esophageal tissues. The presence of peri-esophageal adhesions at operation points toward such a localized abscess and improves the prognosis.

KLAWANS.

Some Questions on the Mechanisms of Action of Bronchoscopy (Quelques questions sur les mechanismes d'action de la bronchoscopie.

Mounier-Kuhn, P., Bronchoscopy, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

The author, feeling that many of the results obtained from therapeutic medical bronchoscopy were due to other factors besides the actual mechanical procedures, studied the reflex mechanisms produced by the entire bronchoscopic procedure—anesthesia, laryngoscopy, bronchoscopy, biopsy, cauterization, etc. He describes changes in pulse and blood pressure which occur during the various stages of the examination and ascribes them to the anesthetic agent, cocain, to vasomotor reflexes, either short, through the nerve plexuses in the bronchial walls, or long through the vagus, and to intrabronchial motor reflexes. He believes that these reflexes are instrumental in producing the so-called atelectasis fruste seen in some foreign body cases.

Accidents such as shock, collapse, and sudden death occurring during bronchoscopy may be exaggerated manifestations of these same reflexes, since these reflexes vary greatly in intensity in different patients and in different regions, and under varying circumstances in the same patient.

KLAWANS.

Bronchoscopic Treatment in Atrophic Laryngo-tracheo-bronchitis (La bronchoscopothérapie dans les laryngo-trachéo-bronchites atrophiques).

Caliceti, P., Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

The author reports favorable results in atrophic laryngo-tracheo-bronchitis, either secondary to atrophic rhinitis or due to syphilis, by bronchoscopy, with removal of crusts by forceps or suction followed by instillation of iodized oil, 25 to 40 per cent. The treatment is repeated as often as necessary.

KLAWANS.

Tetanus Induced by a Foreign Body in the Right Bronchus [Intoxication tétanique provoquée par un corps étranger [anche de trompette] dans la bronche droite).

Tráina, S., and Cilotti, M. (Pisa), Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

The authors report a very rare case of tetanus complicating a metallic bronchial foreign body which had been aspirated twelve days previously. Superior bronchoscopy being impossible because of marked trismus, a tracheotomy was done and the foreign body extracted by inferior bronchoscopy. Intensive serum therapy was then employed, requiring a total of 270,000 units, with eventual recovery.

KLAWANS.

Intratracheal Lipiodol as a Method of Early Diagnosis of Mediastinal Tumors (Le lipiodolage de la trachée. Moyen de diagnostic précoce des tumeurs médiastinales).

Jacod, Maurice, Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

The author reports a case of mediastinal tumor of unknown type and of small size which made it undemonstrable by the usual fluoroscopic and roentgenographic methods. It was well demonstrated when the trachea was injected with lipiodol through an intra-tracheal catheter under the fluoroscopic screen. Endoscopic examination had revealed tracheal compression, but the lipiodol alone allowed the size, shape and location of the mediastinal mass to be made out.

He compares this method to fluoroscopic examination of the esophagus during the passage of a barium bolus and believes that by its use mediastinal tumors too small to give x-ray findings may be discovered and earlier treatment instituted.

KLAWANS.

Difficult Diagnosis of an Esophageal Foreign Body (Diagnostic difficile d'un corps étranger d'oesophage).

Leroux, Louis, Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

The author reports the case of a suspected foreign body in the esophagus which was not found on immediate esophagoscopy but which was found and removed forty-eight hours later. The foreign body proved to be a small piece of bone embedded in meat which in color and texture so resembled the esophageal wall as to be overlooked. Several days later putrefactive changes rendered it visible. After mentioning several other cases in which foreign bodies were overlooked on esophagoscopy, he emphasizes the necesssity for preliminary x-ray studies which, when positive, are of great value, and when negative, are not to be considered as contra-indicating endoscopic examination.

KLAWANS.

Laryngo-Tracheal Compression by Cysts (Compression laryngo-trachéales d'origine kystique).

Vernieuwe and F. Eeman, Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

The authors report three cases of tracheal compression with dyspnea and stridor due to cysts. One was a branchial cyst of large size at the base of the neck, one a cyst in the region of the sinus pyriformis and the aryepiglottic fold, and one large dermoid cyst in the upper mediastinum.

KLAWANS.

Case of Esophageal Foreign Body. Large Denture. Section. Extraction. Recovery (Sur un cas de corps étranger de l'oesophage; volumineux dentier. Section. Extraction. Guérison).

Canuyt, Georges, and Lacroix, Marcel, Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

The authors report the case of a large denture at the lower end of the esophagus which could not be removed at two esophagoscopic attempts because of its large size, irregular shape, and jagged edges which were caught in the esophageal walls. On a third attempt, using a large esophagoscope and a powerful cutting forceps of the authors' own design, the denture was cut into five pieces which were easily removed. This case emphasizes the necessity for a suitable instrumentarium before undertaking foreign body work.

KLAWANS.

Thumb Tack in the Bronchus of a Ten-Year-Old Child for Nineteen Months (Punaise de dessin depuis 19 mois dans les bronches d'un enfant de 10 ans).

Mounier-Kuhn, P., Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January, 1936.

The author reports the case of an unsuspected foreign body in a branch of the right lower bronchus which was extracted only after five bronchoscopies necessitating removal of granulations, drainage of pulmonary suppuration, and dilatation of the stenosed bronchus. The fluoroscope was invaluable in aiding the successful removal of this thumbtack.

KLAWANS.

A Study of Fifty-seven Cases of Bronchogenic Carcinoma.

Rosedale, Raymond, and McKay, Donald (Buffalo), Am. J. of Cancer, 26:493 (March), 1936.

The authors report the essential data of fifty-seven cases of bronchogenic carcinoma recorded in the Buffalo City Hospital from 1924 to 1934. These represented 7.5 per cent of the total number of malignant tumors coming to autopsy during this period, or third in frequency, stomach and uterus heading the list. Seventy-five per cent of the patients were employed in occupations requiring exposure to dust. The tumor was usually unilateral and situated near the lung hilus. Associated pneumoconiosis and tuberculosis was determined at autopsy in fourteen and seven cases, respectively. The authors were unable to divide the histologic material into two groups as Geschickter has done. Clinical diagnosis of primary lung cancer was made in ten of the autopsied cases, suspected in five, and not made in twenty-two. The average duration of symptoms was ten months. Cough was the most constant early symptom. Roentgenograms were

positive for carcinoma in fifteen of thirty cases so studied. Bronchoscopy established the diagnosis in four of six cases done. The authors feel that bronchogenic carcinoma should enter the differential diagnosis of all obscure chest diseases in individuals over 30 years of age. The article is exceptionally well illustrated by charts, photographs and photomicrographs.

JORSTAD.

Bronchoscopy and Broncho-pulmonary Cancers (Bronchoscopie et Cancers broncho-pulmonaires).

Mounier-Kuhn, P., and Piaget, F., Bronchoscopie, Oesophagoscopie et Gastroscopie, Vol. 1 (January), 1936.

Reviewing in a general way the bronchoscopic findings in bronchogenic cancer, the authors urge that exploratory bronchoscopy is indicated in every case of abnormal dyspnea, every unexplained broncho-pulmonary syndrome, every bronchial stenosis with atelectasis, especially in older individuals, and in every case with atypical x-ray findings in the chest. Never make a diagnosis of chronic pneumonia or of asthma without an exploratory bronchoscopy.

KLAWANS.

EAR.

Treatment of Otogenic Meningitis (Traitement des meningites otogènes).

Moulonguet, A. (Paris) and Piquet, J. (Lille), O., R., L. Internat., 19:793 (Dec.), 1935.

Reporting to the French Congress of Otorhinolaryngology, the authors recommend:

1. For septic meningitis with germs in the cerebrospinal fluid, drainage of the cisterna magna with intravenous antiseptics, especially urotropin, and, if time permits, intravenous serum therapy.

2. Meningitis with increased polymorphonuclears but no visible germs; here cistern puncture might spread the infection, hence the operation of Holmgren is recommended, exposing the dura anterior to the sigmoid, over the posterior surface of the petrous, and incising it as low and as far in toward the cerebellopontile cistern as possible; also, when labyrinthitis is present, opening the same region through the internal auditory meatus after labyrinthectomy;

3. "Aseptic" or serous meningitis with leucocytic reaction, usually yielding to repeated lumbar puncture;

4. Hypertensive cortical meningitis, lumbar puncture, with hypertonic intravenous therapy if there be relapse; hypertensive ventricular meningitis may require ventricular puncture. The authors

are afraid of intracarotid injections, pointing out the danger of necrotic changes in the vessel used and also the fact that there is no antiseptic action in the region reached by the vertebral arteries.

Complete and thorough mastoidectomy must necessarily precede any of the above methods, with special reference to the various petrous groups of cells.

FENTON.

MISCELLANEOUS.

Manifestations of Leukemia Encountered in Otolaryngologic and Stomatologic Practice.

Love, Andrew A. (Los Angeles), Arch. Otolaryng., 23:173 (February), 1936.

In the vast majority of cases of acute or chronic leukemia there is clinical evidence of the disease in the ear, nose, mouth and throat. Symptoms of hemorrhage in the middle or internal ear, epistaxis, necrotic lesions in the mouth, throat or larynx; petechial or massive hemorrhages from the mucous membranes of the mouth, the pharynx or the nose or from the gums after the extraction of teeth or any other operation in the mouth, nose, or throat; extreme anemia of the same structures, and necrotic lesions of the larynx or cervical adenopathy without obvious cause all point to the possibility that the patient may be suffering from a blood dyscrasia, especially a form of leukemia. These findings, together with the other general symptoms of this type of disease, indicate the need of an accurate hematologic study.

The bibliography is complete. No work has dealt with this subject completely. Various reports of cases and short dissertations have appeared, however. In view of this, a complete bibliography is appended.

TOBEY.

Tumors of the Oral Mucous Membrane.

Geschickter, Charles F. (Baltimore), Am. J. of Cancer, 26:586 (March), 1936.

Benign tumors are epithelial cysts and adenomas, aberrant salivary adenomas, leukoplakia and benign papillomas. The occurrence of malignancy in leukoplakia is discussed and illustrated. Differentiation may be made only by histologic examination. Relationship of oral hygiene to benign leukoplakia is emphasized.

Carcinoma may begin in an area of keratosis without ulceration or in ulceration without keratosis. In general, carcinoma of the oral mucous membrane is sooner or later characterized by ulceration with a hard raised or papillary edge. Leading clinical features do not favor irritation as a causative factor of carcinoma of the mouth. Ratio of incidence in males and females is ten to one. Duration of symp-

toms of carcinoma of the lip is five years in two-thirds of the cases; of tongue, average under six months; of mucous membrane, 75 per cent, six months to one year; 25 per cent, five years. Treatment was by surgery (usually cautery) and lymph node resection in selected cases. Irradiation was combined in some cases (amount inadequate according to modern conception.) Five year cases were: lip, 395 cases 85 per cent upper, 49 per cent lower; tongue, 15 per cent; mucous membrane, 17 per cent.

In cured cases of carcinoma of the tongue malignancy was of low grade and nodes were not palpable, or there was a small lesion of the anterior third of the tongue without node involvement.

In a similar series reported by Regaud (table inserted), treated by radium, the percentage of cures and number of cases is practically identical. Only three cases of five years' cure were obtained where nodes at carotid bifurcation were involved by carcinoma and these were well differentiated. In tumors accessible to surgery or irradiation there is little or no choice from the standpoint of cures effected. Surgery is preferable to irradiation in treatment of cervical nodes. A judicious combination often gives the best results.

Embryological and endocrine factors must be considered in etiology. Basal cell carcinoma occurred twice as frequently on the upper as on the lower lip.

JORSTAD.

Malignant Tumors of the Upper Face (Les tumeurs malignes du massif facial supérieur).

Portmann, G., and Despons, J. (Bordeaux), Rev. de Lar., Ot., Rhin., 57:1 (Jan.), 1936.

Out of seventy cases, cures were secured in eight out of nine sarcomas, one or two adamantinomas and twenty of fifty-nine epitheliomas. Five of the uncured epitheliomas lived seven years; two others more than five years.

Portmann recommends extensive surgical removal of the growth and all bone attached thereto, followed by massive thermocautery (Percy) rather than diathermic fulguration. He does not use radium or roentgen rays at once or during after treatment unless inoperable recurrences show up. Such tumors are local and rarely metastasize.

Conservation of the eyelids and facial skin, using a lateral nasal incision, has permitted excellent plastic results, although orbital exenteration has often been necessary.

FENTON.